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OUTLINES
OF
GYNÆCOLOGICAL PATHOLOGY
AND
MORBID ANATOMY

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OUTLINES
OF
GYNÆCOLOGICAL PATHOLOGY
AND
MORBID ANATOMY

BY

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WITH 151 ILLUSTRATIONS
MOSTLY ORIGINAL

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PREFACE

My chief object in writing this manual is to describe as simply as possible the morbid processes to which the female organs are subject. I dwell at greater length on those pathological conditions which, owing to their frequency, are of interest to students, and of importance to practitioners.

As far as I have been able to do so, I have tried to keep to the Pathological side of the subject, but I have found it necessary to include certain clinical details, especially in considering the results and sequelæ of the various diseases and conditions described. The anatomy of the subject is also discussed in respect to certain morbid conditions.

I have endeavoured to express the more modern views of British Pathologists, especially those of my teachers at St. Bartholomew's Hospital, to whom I owe so much.

I have taken the greatest care to give references to all authors, both British and Foreign, whose works are quoted in this book. This may also be of convenience to those who wish to go further into any special subject, or to students who wish to look up particular papers for examination purposes or other reasons.

The drawings with which this work is illustrated are mostly original. The greater number have been drawn by myself from the actual specimens, either in various museums, or from material placed at my disposal at the Samaritan Free or

St. Bartholomew's Hospital. Each specimen has also been photographed and my drawings finally reproduced with the greatest care by Messrs. J. P. Collings and Sons, who have expended much time and trouble over them, in endeavouring to obtain in each case a faithful and exact reproduction, and not a mere diagram.

Each drawing is accompanied by a reference to the place where the original may be found, so that, if necessary, students may refer to what the Author hopes are more or less representative specimens of the subject.

Some pictures have been borrowed from other sources, or blocks have been lent by the kindness of other writers on the subject, to whom the Author wishes to express his grateful thanks.

They are acknowledged in each case.

Dr. Cullingworth has allowed me to make drawings of many of the specimens already illustrated in his work on 'Diseases of the Fallopian Tubes.' Dr. Herbert Spencer has permitted me to reproduce some beautiful drawings of 'Deciduoma Malignum.' For other pictures I am indebted to Dr. Griffith, Dr. Amand Routh, Dr. Walter Tate, Dr. Eden, Dr. Giles, Mr. Alban Doran, Mr. Bland-Sutton, Mr. Targett, Mr. J. S. Taylor, and Sir W. M. Banks.

The micro-photographs have been prepared by Dr. C. H. Cosens, who has devoted much valuable time to the subject. They are taken from actual slides prepared by myself, or from others kindly lent me by Mr. Corrie Keep.

My thanks are due to the Curators of the Museums of the Royal College of Surgeons, St. Thomas's Hospital, and St. Bartholomew's Hospital, both for permission to reproduce the specimens and for their courtesy in helping me with my work.

To the Council of the Obstetrical Society of London

I am much indebted for permission to reproduce some of the drawings in the *Transactions*.

Ectopic Gestation has been included, owing to its important pathological bearings, and brief reference is made to Primary Ovarian Pregnancy. In adding a short chapter on Micro-organisms, the Author has largely followed Muir and Ritchie, Bowlby, and Kanthack. Dr. Drysdale has helped me very much in this subject. 'Cancer Parasites' are shortly discussed.

Mr. Alban Doran has kindly given me both his valuable assistance and advice in revising the proof sheets.

C. HUBERT ROBERTS.

21 Welbeck Street, W.
September 1901.

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GYNÆCOLOGICAL PATHOLOGY

CHAPTER I

ON DISEASES OF THE VULVA

Age Changes.—In infants, the labia majora are merely rounded parallel firm cutaneous folds; the nymphæ are very thin and project between them, and are very small in comparison with the labia majora; both the mons Veneris and labia are devoid of hair.

At puberty, hair grows on the mons Veneris and outer surfaces of the labia, while the labia majora themselves increase in size, and are in apposition, covering the nymphæ, which are bright pink in colour.

At the menopause and during old age, the hair becomes grey or white, and may be shed; the labia shrink and atrophy, exposing the nymphæ and vaginal orifice; the vascularity of the parts is much diminished, the pink colour lost, the surfaces become pale and smooth, and the orifice is narrowed.

Malformations of the Vulva.—The most interesting defects are those seen in cases of so-called Hermaphroditism, and in the deformities in which the cloacal condition is approached.

Absence of the Vulva.—This, according to Ballantyne,[1] occurs only in non-viable fœtuses, chiefly of the acephalic and sympodial types; the skin passing straight from symphysis to coccyx.

Internally (but without any opening externally), the cloaca or sinus urogenitalis may exist.

Superficial Vulval Atresia is probably due to adhesive vulvitis, which leads to a glueing together of the labia; it may be present at birth, or be developed in infancy: generally the occlusion is not complete, and a small orifice exists below the clitoris through which urine escapes.[1]

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[1] Ballantyne, Allbutt and Playfair, *System of Gynæcology*, art. 'Malformations of the Genital Organs,' p. 91, and complete list of references.

The adhesions can easily be broken down.

An **infantile** condition of the vulva may exist. It is important, as it may indicate such associated defects as deformity of uterus or ovaries.

Abnormal communications of the Vulva.—The most interesting of these conditions is persistent cloaca; here apparently the normal anus is absent, and by imperfect growth downwards of the perineal partition, the rectum opens into the urogenital sinus (not the vagina really,[2] for the Müllerian ducts have not yet grown downwards to form the lower part of the vagina).

Hypospadias in Women. (Persistent Urogenital Sinus.)—Here the urethra opens into the vagina, or more strictly speaking the urogenital sinus, and through the common opening, which is situated at the base of the clitoris, both urine and menstrual fluid escape. The perineum is normally formed, and the rectum opens separately behind it at the anus; thus differing from the cloacal condition described above. Sometimes the clitoris is hypertrophied and prominent. In another variety there is absence of the urethra and the vagina, and the bladder opens into the vestibular canal,[3] with incontinence of urine, and it appears on inspection as if the bladder opened directly into the vagina.

Epispadias in Women.—Defect of the upper wall of the urethra may occur alone, or with malformations of the bladder and anterior abdominal wall.

If the urethra only be defective above, it exists as an open groove passing upwards to the vestibule; the clitoris may be split, with the labium minus attached to each half. The labia majora may or may not diverge, the bladder is closed but small, and the symphysis does not gape. Ballantyne[4] quotes a case, seen by himself, in which there was defective growth of the pubic hair.

The most severe forms of epispadias are complicated by extro-version of the bladder, and the pelvis is split; the symphysis gapes, the labia majora are wide apart, and urine escapes from the exposed bladder directly from the ureters. Sometimes a cloacal condition exists as well.[1] Epispadias is much rarer in the female than the male. (See also 'Ectopia Vesicæ.')

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[1] Ballantyne, *op. cit.* p. 92.

[2] *Op. cit.* p. 93.

[3] *Op. cit.* p. 95.

[4] *Op. cit.* p. 95.

Malformations of the Clitoris and Labia.—The clitoris is sometimes absent in epispadias, or it may be split; further, the clitoris may be rudimentary or there may be hypertrophy.

These cases are of interest, as if combined with labial herniæ the individual resembles the male type.

The labia are sometimes enlarged or glued together. Sometimes the labia minora are increased in size, and form two large pendulous folds protruding between the labia majora.

Imperforate Hymen.—Probably a few undoubted cases do occur of atresia of the hymen, but in the majority of recorded cases the atresia is really vaginal, and, as Matthews Duncan [2] points out, the normal perforate hymen can be seen in its usual situation, or bulged forwards and spread out on the surface of the sac formed by the occluded and distended vagina.

The results of this are considered in relation with Retention of the Menses.

Anomalies of the Hymen.—These have practically little importance; the orifice may be exceedingly small and difficult to find, or be circular or linear, or there may be more than one orifice; the hymen may sometimes be abnormally thick and rigid, or very vascular.

HERMAPHRODITISM

In the old days the term hermaphrodite was applied to individuals who were supposed to possess the perfect organs of both sexes. No such thing exists in the human species. Modern authors have come to use the word rather to indicate subjects of doubtful sex.

As Ballantyne [3] points out, at one stage of its existence the human embryo may be regarded as an hermaphrodite, and it is easy to understand how morbid influences brought to bear upon the embryo at, or about, the time when it is passing from its sexually indifferent stage into one of differentiation, may so upset the normal process of development as to produce an individual with, for example, testicles and a uterus. It is, however, a matter of great difficulty to imagine a condition of affairs which would give rise to the presence of a testicle and an ovary on the same side, for the sexual gland may become an ovary or a testicle, but not both; and it is doubtful whether a genuine [1] case of the co-existence of well-developed testicles and ovaries in the human subject has ever been reported,[2] but instances of Pseudo-Hermaphroditism are far from rare. By Pseudo-Hermaphroditism is meant that, along with either ovaries or testicles, there are found some of the genital organs of the opposite sex.

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[1] Ballantyne, *op. cit.* p. 96.

[2] Matthews Duncan, *Clinical Lectures*, 4th ed., p. 108.

[3] Ballantyne, *op. cit.* p. 100.

The greatest care must be taken before it can be certain that swellings supposed to be ovaries or testicles are really such; no real opinion should be given unless such swellings have been examined microscopically.[3] This is referred to again in Hernia of the Ovary.

Pseudo-Hermaphroditism.[4] — This means that although the genital glands are of one sex only, the excretory ducts of both sexes are present, and the individual appears to possess the organs of both sexes, although this is really not the case.

The subject of these malformations may be either male or female; the malformations vary in degree, and each kind of Pseudo-Hermaphroditism (male and female) has been divided into internal, complete, and external.

Male Pseudo-Hermaphroditism[5] is much commoner than the female type. The following varieties exist:

a. **Internal.**—Where there are testicles in association with external genitals of the male type, and a uterus, vagina, and even Fallopian tubes.

b. **External.**[6]—In this variety there are also testicles, but the external genitals and the build of the body are feminine. This variety is common.

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[1] Herman, *op. cit.* p. 614.

[2] See Blacker and Lawrence's case and criticisms, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 265; and Klebs, *Handbuch der Path. Anat.* vol. i.

[3] Bland-Sutton, *Diseases of Ovaries and Tubes*, 1891, p. 28.

[4] Ballantyne, Allbutt and Playfair, *System of Gynæcology*, p. 102, with literature, p. 111; and Herman, *Diseases of Women*, p. 614.

[5] Ballantyne, Allbutt and Playfair, *op. cit.* p. 102.

[6] Herman, *op. cit.* p. 615.

c. **Complete.**—In this there are testicles, but there is also a uterus masculinus with Fallopian tubes, and the external organs approach more or less closely the female form.

In **Female Pseudo-Hermaphroditism** there are always ovaries; three varieties also exist.

a. **Internal.**—Here ovaries exist, but there are also distinct traces of Wolffian ducts.

b. **External.**—Where ovaries exist, but the external genitals are of the male form.

c. **Complete.**—Here ovaries occur, the external organs are masculine, and Wolffian ducts and prostate gland are present.

One of the most usual arrangements of parts to which the name of Female Pseudo-Hermaphroditism is given is that in which a woman has adhesion of the labia, with hypertrophy of the clitoris, and labial ovarian herniæ.

Pseudo-hermaphrodites are generally sterile,[1] for the organs which resemble those of the opposite sex are generally badly developed.

Non-descent of the testicles, and the existence of scrotal hypospadias, or a small imperforate penis (the urethra opening at its root, together with a sort of vulvar aperture), together with a hymen, makes the resemblance of such a male to a female very striking.[2]

Enlargement of the breasts in the male, and the absence of hair on the face and chest, make it still more difficult to determine the sex of such cases.

Ballantyne thinks that most of the cases of supposed true hermaphroditism are really cases of hypospadic males; and Herman[3] advises that if there be doubt as to the sex of an infant, and if pressed for an opinion, advise that it be brought up as a boy. Lawson Tait makes the same rule.

Cases of Pseudo-Hermaphroditism may occur in the same family.[4] Mental and moral weakness, and even insanity, may occur with this condition.

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[1] Herman, *op. cit.* p. 615.

[2] Ballantyne, *op. cit.* p. 103, and Targett, *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 272; also E. Willett, *Trans. Path. Soc. Lond.* vol. xlv. 1894, p. 102.

[3] Herman, *op. cit.* p. 615.

[4] Ballantyne, *op. cit.* p. 105, who quotes J. Phillips's and Lindsay's cases.

Vulvitis.—Inflammations of the vulva are difficult to classify; the following varieties may, however, be recognised:[1]

1. Traumatic.
2. Catarrhal.
3. Dermal.
4. Ulcerative.
5. Septic.
6. Diphtheritic.
7. Erysipelatous.

Traumatic Vulvitis generally arises from some injury, scalds, or the application of caustics. Injuries of the vulva are important, especially in relation to childbirth;[2] more will be said about them in speaking of rupture of the perineum.

Wounds of the vulva[3] in the pregnant condition are liable to be attended with serious or even fatal hæmorrhage, as the parts are extremely vascular.

Any wound of the vulva may be the source of entry of septic organisms, and all such lacerations following parturition should, if possible, be closed at once.

Catarrhal Vulvitis.—This may be acute or chronic; it includes a very large number of cases of inflammation of the vulva, and occurs at all periods of life; in fact, a classification of vulvitis might be made, based on the various ages at which such discharges and inflammations occur. Generally there is some more or less constant source of irritation.

Catarrhal Vulvitis in Infants and Children.—This is usually due to some want of cleanliness, decomposing urine or fæces, or irritating applications, or even diapers which have been washed in strong soda; in such cases the surrounding parts are also affected.

Worms are frequently quoted as a cause of infantile vulvitis, but on rather doubtful evidence. A few cases are associated with tuberculosis; in others, the bacillus coli has been found in the discharges.

The vulvitis of children[4] is frequently ascribed to gonorrhœa, but it is quite certain that purulent vulvitis and vaginitis may exist in children quite apart from this cause.[5] This question is obscure; the mere discharges prove nothing; the gonococcus of Neisser, if present, proves something; the question is whether it can be always identified in conjunction with other micro-organisms. Statements on the etiology of purulent vulvitis in children are at present confusing, but a large proportion exist which are of a simple nature, and can be cured by simple methods. The onus of proving that they are gonorrhœal rests with those who make the statement, and in practice it would be unwise to do so unless one is sure. Vulvitis in children is rarely associated with vaginitis.[1]

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[1] Smyly, Allbutt and Playfair, p. 372.

[2] Herman, *Morbid Conditions of the Female Genital Organs resulting from Parturition*, Allbutt and Playfair, p. 425.

[3] Dakin, *Handbook of Midwifery*, p. 587.

[4] Drummond Robinson, 'Vulval Discharges in Children,' *Trans. Obstet. Soc. Lond.* vol xli. 1899, p. 14.

[5] McCann, 'Gonorrhœal Infection,' *Encyclopædia Medica*, vol. iv. p. 196.

Vulvitis in Adults.—This is commonly caused by dirt and uncleanliness, putrid vaginal discharges of all kinds, either local, or coming from other parts of the genital canal. Such discharges may be due to cancer of the cervix, sloughing fibroids, decomposing products of conception, ammoniacal foul urine, vesico-vaginal fistula, foreign bodies in the vagina, septic infection after delivery, and venereal infection.

Gonorrhœa, as a cause of purulent vulvitis in adults, is important, but not all cases are gonorrhœal that have a purulent discharge.

The chief importance of gonorrhœa is its relation to pelvic inflammation.[2] In gonorrhœa there may be few local symptoms to attract attention, and the earlier symptoms are disregarded, as there is little pain, unless the urethra be involved, and advice is sought late. In women, gonorrhœa is a more serious disease than syphilis.

The most frequent lesion in gonorrhœa is inflammation of the ducts of the vulvo-vaginal glands and the parts around. Urethritis is common. There is often swelling, œdema, and ulceration of the nymphæ and labia majora. The vagina is rarely affected, but the infection of the mucous membrane and glands of the cervix is common, and very important, chiefly for the reason that infection there is difficult to reach and to treat, and the endometrium, tubes, and peritoneum are sooner or later involved. The tubes become sealed, pyosalpinx is common, and the patient suffers from more or less acute and recurrent attacks of pelvic inflammation. Generally, the peritonitis is limited by adhesions.

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[1] See chapter on 'Gonorrhœal Infection in relation to Pelvic Inflammation.'

[2] Cullingworth, *Brit. Med. Journal*, July 20, 1889.

Nœggerath[1] has also pointed out the importance of gonorrhœa in relation to diseases of women.

Sänger[2] states that 12 per cent. of all women who consult a gynæcologist suffer from gonorrhœa, and that over one-third of sterile marriages are due to this disease; and further, that acquired sterility after the birth of one child is, as a rule, due to gonorrhœa. Epidemics of vulvo-vaginitis in schools may be due to this cause.[3]

In acute vulvitis, from whatever cause, there is generally muco-purulent or purulent discharge, the vulval structures are red and swollen, and there is often œdema; the glands of Bartholin are involved and abscess results; urethral infection in gonorrhœa is common.

Local excoriations are sometimes found. A one-sided œdema of the labium should suggest some local source of infection. The acute septic inflammations of the vulva will be described later.

In **Chronic Vulvitis** there is generally less swelling and excoriation, glandular structures may be involved, pustules and follicles inflame, and there may be local hypertrophies; shallow excoriations sometimes exist over the labia minora and vestibule (possibly the 'lupus minimus' of Matthews Duncan[4]).

Nearly all these cases are associated with some chronic discharge which keeps up the irritation, and such must be investigated and cured before the local vulvitis will yield to treatment.

Dermal Vulvitis in a simple form is seen in very stout women, amid the various folds of the vulva, thighs, and abdominal walls. Another variety is due to eczema in a chronic form, and some cases of 'Pruritus Vulvæ' are of this nature.

Herpes of the vulva is sometimes seen; it is characterised by the appearance of vesicles, pustules, and scabs in groups or crops. It may occur in pregnancy; more rarely the herpetic attacks take place at each menstrual period.

Prurigo, in which small papules appear, causing the most distressing itching, is attributed by Klebs to dilated lymphatics in hypertrophied papillæ.[5]

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[1] *Die latente Gonorrhöe im Weiblichen Geschlecht*, Bonn, 1872.

[2] M. Sänger, *Verhandl. der Deutschen Gesellsch. f. Gyn.* Leipzig, 1886.

[3] Smyly, Allbutt and Playfair, p. 373.

[4] Matthews Duncan, *Clin. Lect.* 4th ed., 'On Lupus of the Pudendum,' p. 201.

[5] Smyly, Allbutt and Playfair, p. 377.

Inflammations of the sebaceous follicles are seen in chronic vulvitis, and pustules may develop. The condition is allied to acne, the glands become distended with their own secretion, and suppuration occurs.

Follicular Vulvitis,[1] as described by Huguier, is generally associated with pregnancy. It is a severe form of general dermatitis, accompanied by pustules and papules on the labia, swelling, induration, and ulceration of the parts, together with irritation and offensive discharge. It appears to exist mostly in the lower orders abroad, and to have some relation to syphilis. It is rare in England.[2] Another form described by Herman[3] consisted of deep red spots about the size of a pin's head scattered on the surface of the nymphæ. Gonorrhœa was probably the cause, but not certainly so.

Ulcerative Vulvitis.—This generally occurs in young children after the exanthemata; small ulcers appear which may become gangrenous.

Noma Vulvæ occurs in children generally after infectious, febrile, or wasting diseases, such as measles, scarlet fever, typhus, and phthisis. It probably depends upon a special microbe. Generally one labium is affected, there is a dark livid spot with induration, vesicles or bullæ appear and superficial sloughing takes place. Finally, gangrene occurs, which may destroy the labium and spread to surrounding parts, such as the vagina, or mons Veneris.

Gangrene of the Vulva in Adults.[4]—This condition is rare; it may occur in the course of acute specific diseases such as small-pox and typhus, or as an acute septic puerperal infection with rapidly spreading destruction of tissue. Another form of gangrene seen in septic cases begins as bilateral inflammation of the labia with the formation of a slough; this may extend to the perineum, anus, and lower part of vagina and urethra. Rapidly spreading gangrenous erysipelas may affect the vulva and tissues around; sometimes in severe puerperal cases a similar sort of cellulitis occurs with great induration spreading from the vulva.

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[1] Herman, *Diseases of Women*, p. 478.

[2] Matthews Duncan, *Clin. Lect.*, 4th ed. p. 182.

[3] Herman, *op. cit.*, p. 480.

[4] Herman, *op. cit.*, p. 494, and *Trans. Obstet. Soc. Lond.* vol. xxv. 1883, p. 141, 'Case of Acute Gangrene of the Vulva in an Adult.'

Septic Vulvitis includes many of the cases described above as puerperal, such cases generally occurring after parturition. They are attended with œdema, swelling, and ulceration, which may even assume a gangrenous type. One form of this occurs as the so-called 'puerperal ulcer'; here one labium is found œdematous, and when examined on the inner surface, a fissure or ulcer can be discovered, having a white base with a thin irritating fœtid discharge, the ulcer being surrounded by inflamed red indurated margins. If the ulceration be very severe, and much tissue be destroyed, recovery may be followed by stenosis of the vulva.

Diphtheritic and Dysenteric Vulvitis are complications of the two diseases respectively concerned. Care must be taken to be sure that mere membranes or sloughs found in inflammations and ulcerations of the vulva are really of such a nature, before the diagnosis is formed. Such conditions are very rare. Erysipelas of the vulva may, however, occur.

Pruritus Vulvæ.—Pruritus means 'itching' of the vulva, and as such is a symptom rather than a disease; it is a common complaint; it may be slight and transitory, or most severe and distressing, the irritation becoming unbearable.

The irritation is often worse at night when the patient is warm in bed, nor is the itching necessarily confined to the vulva, but surrounding areas may be affected, such as the anus, thighs, or the front of the abdomen and mons Veneris. The itching in bad cases is so intolerable that nothing will prevent such patients from attempting to obtain relief by scratching, and in some cases, so miserable is their condition, that they have committed suicide, or become insane, on account of this.

Locally, in pruritus, the vulva is found excoriated and inflamed, in some cases dry and cracked, with little fissures which easily bleed, and everywhere marked with scars and scabs caused by the constant scratching; in others, the surrounding skin is reddened, the labia majora thickened, covered with a thin irritating secretion, or even with a moist wash-leathery-looking macerated skin.

Great care must be taken in such cases to distinguish between cause and result, for much of the condition found may be the result of the patient's continued scratching. The same remarks apply to microscopical examination of pieces of skin removed from the affected areas. Such changes have been described by Webster[1] and Sänger;[2] the changes observed were those of chronic inflammation. Exudation of inflammatory cells and organisation of fibrous tissue, special changes in nerve-endings, Meissner's capsules and Krause's genital corpuscles, are described, due to fibrosis, or pressure from inflammatory connective tissue; fibrosis is also said to affect the nerves of the vulva. Webster[3] thinks the fibrosis of nerves distinct from chronic inflammation, and that it is a special process (dermato-neuritis) found in Pruritus.

Causes of Pruritus.—A classification of the causes of pruritus is difficult.

Sometimes no cause can be found, and these may be designated **Primary,** while those in which there is some evident irritating discharge may be termed **Secondary.**

The primary cases come under the heading of neuroses; such cases occur in women about the menopause, very rarely in young women, but in these each menstrual period may bring on an attack of pruritus. Pregnancy, is sometimes associated with this condition.

Diabetes and Pruritus.—In pruritus the urine should always be examined for sugar. There may be either glycosuria or diabetes present. It has been thought that it was the contact of saccharine urine that caused the vulvitis, but the areas affected by the pruritus of diabetes do not always coincide with those moistened by the urine. Glycosuria brings with it a tendency to inflammation of the skin. In the majority of cases the itching precedes the inflammation of the vulva, and pruritus may be an early symptom in diabetes. Sometimes there are local changes of the vulva, either dry scaly inflammation with reddening, or it may be moist with much discharge. Possibly diabetes and neuritis are intimately allied, such neuritis, rather than the irritating urine, being the cause of pruritus. In well marked cases of diabetes with a large quantity of urine, there is sometimes no pruritus at all.[4] Howard Kelly regards the condition as due to trophic lesions, not to the contact of urine; excision of the local areas of the vulval skin affected by such changes is said to cure the irritation.

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[1] C. Webster, *Lab. Reports R. Coll. Phys. Edin.*, 1891.

[2] Sänger, *Centralb. für Gyn.*, 1894; also C. Webster, *Diseases of Women*, p. 551.

[3] C. Webster, *op. cit.* p. 551, figs. 209, 210.

[4] Herman, *Diseases of Women*, p. 470, quoting Fagge.

Secondary Pruritus includes those cases of itching to which some definite local cause can be assigned. Any irritating discharge will do this, so that pruritus may occur with gonorrhœa, senile endometritis, cancer, sloughing fibroids and polypi, foreign bodies in the vagina, irritating cystitic urine, or where there is an escape of urine or fæces over the vulva, as in vesico-vaginal or recto-vaginal fistula. Pediculi and worms are also given as causes ; it is possible the former might cause irritation. Dirt and retained secretions may act in the same way.

Some of the cases termed pruritus are really true cases of eczema, of an alcoholic or gouty nature, and the condition is really a dermatitis ; others again depend upon irritation not of the vulva but of the anus, in connection with hæmorrhoids and fissure, the itching being sometimes very distressing.

Urethral Caruncle is described in the chapter on Diseases of the Urethra.

Chancre.—The true Hunterian chancre affects the vulva. It is hard, though its hardness is difficult to feel.[1] Generally, a chancre, if it be on the skin surface of the labium, has the ordinary characters of a hard sore, usually covered by a crust with very little discharge. The inguinal glands are affected and are very hard, while the labium on which a chancre is situated is frequently œdematous. If the chancre be on the mucous surface of the labium, it looks like a small fissure with a coppery indurated area round it, but it is difficult to pick up the tissues around to feel the induration. True Hunterian chancre may occur on the cervix,[2] but is very rare. Occurring in this situation it has a mottled yellow ecchymosed appearance, not the uniform red surface of an erosion, and has a broad zone of congestion around it.

Soft Chancres are as a rule multiple and symmetrical, for they can inoculate themselves ; the sore generally has a roundish appearance, and a surface like wet wash-leather, with an inflamed area around. It may be covered with a scab. The inguinal glands are affected and may suppurate. The glandular enlargement is never as hard as that due to the Hunterian chancre.

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[1] Herman, *Diseases of Women*, p. 481.

[2] Herman, *Trans. Obstet. Soc. Lond.* vol. xxvii., 1885, p. 252, plate 15.

Syphilitic Condylomata are commonly seen as flat reddish or greyish-white raised patches with a more or less sinuous outline; they often coalesce, and the discharge may be offensive. Vulval condylomata generally co-exist with similar typical patches on the perineum and around the anus. A rash is frequently present on the body. Condylomata occur early in secondary syphilis.

Esthiomène, or so-called Lupus of the Vulva.—This was described first by Huguier[1] and the name esthiomène (eating) given to it. Matthews Duncan[2] called it lupus, and apparently regarded it as identical with lupus elsewhere, but he says that in the sections examined by Thin the disease is histologically unlike ordinary lupus, the morbid structure being diffused in the affected parts, not in nodules or tubercles.

Sometimes it is a syphilitic disease, and this view is held by Hutchinson. Matthews Duncan said that often there was no syphilitic history, and that women affected are 'often fine, healthy, even blooming in appearance. The disease has a peculiar history, and such extraordinary changes or transformations as to separate it from every other.'[3] The main features of this disease are ulceration and overgrowth; the ulceration generally affects the vulva, clitoris, vestibule and urethral orifice, or may spread to the mons Veneris, perineum, and rectum. The ulcers secrete pus; they may cover a great area, or they may burrow far and wide, and cause destruction of the whole ano-perineal region; the urethra, vagina, and rectum may be involved, but they never attack bone. The ulceration may stop spreading, and in time it may heal, or it may heal at one place and spread at another. Matthews Duncan says these ulcers may bleed dangerously.

Hypertrophy, the other change so typical of esthiomène, is constant; there is always more or less overgrowth, which affects the same parts as the ulceration. These growths consist of fibrous indurations of the skin, mucous membrane, or subcutaneous tissue; in some cases they are pedunculated and irregular; in others, the enlargement may be a general one of the labium or clitoris. The overgrowths may vary in colour and appearance, or they may inflame. Contractions may take place in esthiomène leading to stricture or occlusion of the genital canal. Herman[1] thinks that tubular stricture of the rectum in women is of a similar nature.

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[1] *Mém. de l'Acad. Nationale de Méd.* vol. xiv. 1849, quoted by Herman, *Diseases of Women*, p. 485.

[2] Matthews Duncan, *Clin. Lect.* 4th ed. p. 201, 'On Lupus of the Pudendum.'

[3] Matthews Duncan, *op. cit.* p. 203.

Matthews Duncan[2] says that this lupus or esthiomène is a different thing from the so-called elephantiasis, seen in tertiary syphilis, nor has it any connection with true elephantiasis.

Kraurosis Vulvæ. (Drying, Hardening.)—This is a rare condition, and is characterised by peculiar shrinking of the skin of the external genitals, attended with great tenderness, and with atrophy and contraction of the vulval orifice.

Briesky[3] of Prague first described it in 1885, as a progressive atrophy of the nymphæ and vestibule. The writer has not seen Briesky's paper. Herman[4] also says the paper appears in a journal inaccessible in London. Martin[5] and Orthmann[6] also describe it. Lawson Tait[7] seems to describe a similar condition, and as far as the description goes it appears to be the condition not infrequently met with in women about the climacteric; Briesky's cases were in connection with pregnancy.

Climacteric dyspareunia, Herman[8] says, is a suspicious symptom of this condition.

In Kraurosis, the tissues of the vulva are smooth and pale, with irritable tender red patches along the margins of the hymen and in the vestibule, which are apparently subcutaneous hæmorrhages. The urethral orifice is caruncular, red, and tender. The tissues later lose their elasticity and become dry, and assume a whitish appearance as if they had been smeared over with strong carbolic acid. Sometimes there are deep cracks or fissures in the labia or near the fourchette, which bleed on any attempt at examination and are very painful; in other places the epithelium is thick or heaped up into little nodules, and the hairs on the labia are coarse and broken. The pubic hair is short or more or less absent. Atrophic changes follow, and the labia disappear or are smooth and very pale, with

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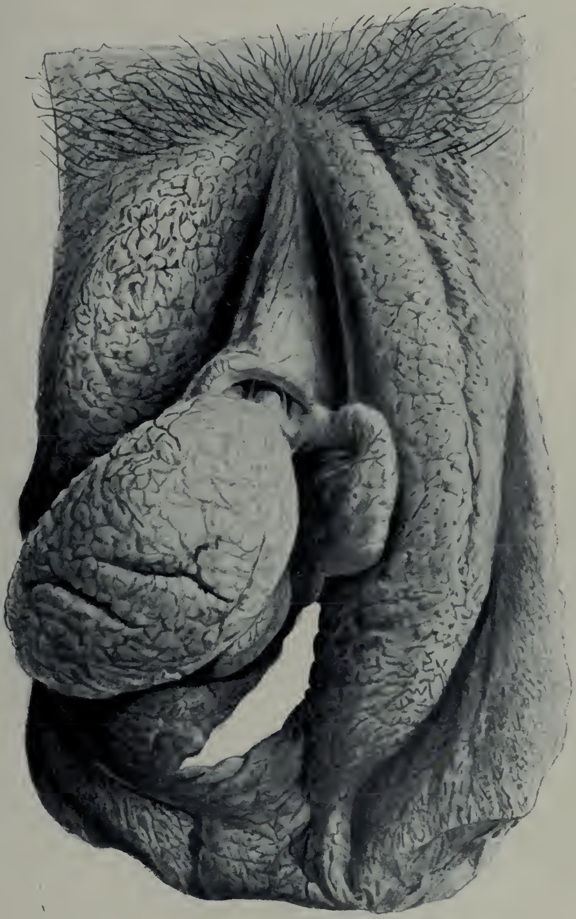
[1] Herman, *Diseases of Women*, p. 488.
[2] *Clin. Lect. Dis. of Women*, p. 205.
[3] Briesky, *Zeit. f. Heilk.* vi. 69, 1885.
[4] Herman, *op. cit.* p. 621.
[5] Martin, *Volkmann's Sammlung*, 1894.
[6] Orthmann, *Zeit. f. Geb. und Gyn.* vol. xix. 1890, p. 283.
[7] Tait, *Diseases of Women*, vol. i. p. 44.
[8] *Diseases of Women*, p. 622.

FIG. 1.

ENLARGEMENT OF THE NYMPHÆ (SO-CALLED ELEPHANTIASIS).



The external organs of generation of a woman, showing irregular coarse enlargement of the nymphæ and neighbouring parts. (This is not true elephantiasis.)

(Specimen 4679. Museum, Royal College of Surgeons.)

no folds or creases, and the orifice of the vagina becomes so rigid and contracted that it will not admit a finger. There may be great irritation (pruritus), accompanied by discharge, and the tissues are very friable. The vagina, except at the orifice, is not affected; the uterus is generally small, but it is so only by senile atrophy, and does not seem to be affected by the disease.

Nothing is known of the cause of Kraurosis; it is probably not syphilitic or gonorrhœal; it generally occurs in elderly women. Scleroderma has been compared to it. Herman[1] likens it to Dupuytren's contraction of the palmar fascia.

Microscopically, there is atrophy of the corium and great increase in small cell infiltration and fibrous tissue parallel to the surface, followed by contraction. The glands of the vulva disappear, vessels and nerves are said to be compressed, and the rete Malpighii is so thin that the epidermis rests directly on the papillæ, which are small, and very sensitive at first. Later, the pain disappears and the parts are insensitive. The disease is chronic, and nothing gives much relief.

Excision of the affected area seems to do some good; it certainly relieved a patient operated on by the author at the Samaritan Free Hospital, but it may recur.

Elephantiasis.—This condition is seldom seen in England, and appears to be essentially tropical in origin. The condition must not be confounded with mere overgrowth and hypertrophy of the labia or clitoris, which may be termed Spurious Elephantiasis. (Fig. 1.)

True Elephantiasis depends upon the blocking of the lymphatics by the Filaria sanguinis hominis; these can be found in the blood early in these cases, but not later on, as the lymphatics are blocked by the solid fibrous enlargement of the parts.

The disease is characterised by great enlargement of the labium or clitoris, and, unlike esthiomène, there is no ulceration. The growth is very slow but may attain to very enormous dimensions, the surface may be irregular, nodular, warty (elephantiasis verrucosa), or even papillomatous (elephantiasis papillomatosa), and in the interstices decomposition of secretions takes place. In other cases the surface is smooth and shining (elephantiasis glabra). The surface may become ulcerated or gangrenous from injury. Sections of the tissues show blocked and dilated lymphatics. In large tumours the blood-vessels are of great size. The urethra may be curiously distorted in tumours originating from the clitoris. The legs may be affected in true elephantiasis.

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[1] Herman, *op. cit.* p. 623.

Howard Kelly[1] believes that elephantiasis of the vulva is distinct from that which affects the lower extremities, the hypertrophy being due to inflammation associated with obstruction to lymph channels draining the external genitals; and that syphilis is one of the commonest exciting causes.

It is probable that the word 'elephantiasis' is applied to various forms of enlargement of the vulva, whether due essentially to the filaria or to other conditions causing hypertrophy.

Local Hypertrophy of the Clitoris may occur; in other cases the **labia minora** are sometimes enormously enlarged and project at the vulval orifice, becoming altered in texture. This is sometimes congenital; in others, it is the result of inflammation and œdema.

Cancer of the Vulva.—This is not a very common disease, and is much rarer than cancer of the uterus. It occurs chiefly in old women, and may begin either in the labium or clitoris; the usual form is a squamous-celled epithelioma (Fig. 2), and it is the analogue of so-called 'sweep's cancer' of the scrotum.

The growth, which is often preceded by irritation and leucoplakic patches, may begin as a small nodule, which is pale and hard; it spreads slowly, but eventually its surface ulcerates, such an ulcer having raised hard borders. The surface is a little depressed and is unhealthy, with areas of slough and ulceration which look whitish or grey, covered with a foul thin discharge. Epithelioma does not spread rapidly, but it spreads surely, gradually involving the whole labium; it tends to spread mostly inwards towards the mucous surface, and not outwards; sometimes there is deep ulceration and great pain. The inguinal glands are involved, and may be much enlarged and very hard.

Sometimes the disease is bilateral, or rather, the growth on one labium seems to implant itself upon the opposite one. The author has seen one such case; Howard Kelly[2] gives an account of another case of the same sort, and also quotes one

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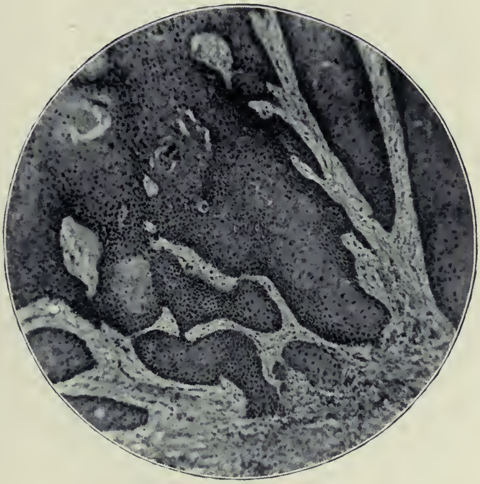
[1] *Operative Gynæcology*, vol. i. p. 181.

[2] *Op. cit.* vol. i. p. 175.

Fig. 2.

EPITHELIOMA OF THE VULVA.

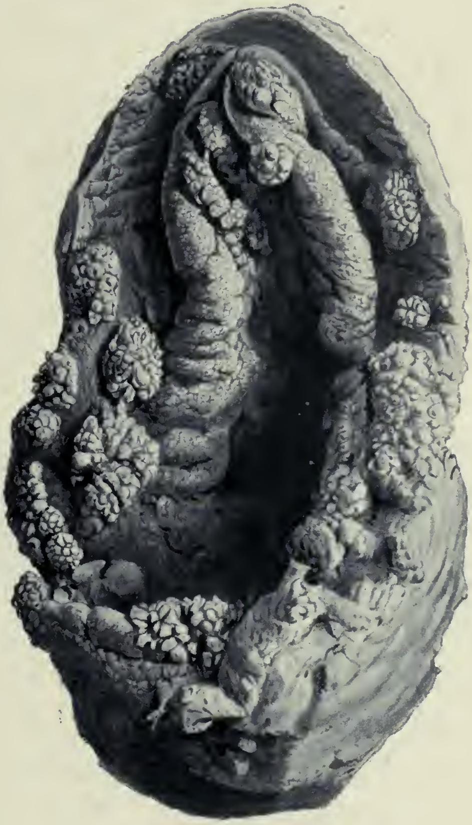


Section of a malignant growth which sprang from the labium majus of an old woman. It shows well-marked invasion of the stroma from above downwards by columns of squamous epithelial cells, which in places contain cell-nests. There still remain a few long branching processes of the deeper layers of the original tissue.

(Microphotograph by C. H. Cosens.)

Fig. 3.

WARTS OF THE VULVA.



The external organs of generation of a woman which were removed post mortem. The labia majora, labia minora, and the region of the vestibule are everywhere studded with warty growths which are almost symmetrically placed.

(Specimen 4689. Museum, Royal College of Surgeons.)

in which the implantation occurred with cancer of the clitoris.

Cancer of the Clitoris occurs as often as cancer of the labium, oftener according to Herman[1]; it is generally a sharply defined, granular, warty, reddened growth, often very painful; the growth is generally a squamous epithelioma with typical cell-nests, accompanied by areas of epithelial cell-invasion. The vessels leading to the growth are in many cases very large.

Melanotic Sarcoma is a curiosity when it occurs in the vulva. The tumour has the typical bluish black colour. Its growth is very rapid; the nearest lymphatics are involved very early.

Cancer of the Urethra may eventually spread outwards and involve the tissues of the vestibule (see Diseases of the Urethra).

True Carcinoma of the Vulva is very rare. The cases reported appear to have started in Bartholin's gland.

Warts may begin anywhere on the vulva (Fig. 3), and may spread to the skin around the perineum, anus, and vagina. They start in small scattered areas as minute papillomata; eventually huge masses may be found, with a typical lobulated appearance, bathed in very foul secretion from the decomposition which goes on in the deep interstices of the mass. Hæmorrhage may take place from the surface. Some of these masses are more or less sessile, others pedunculated, and are generally bilateral, as if implanted from one side to the other. They appear to be due to any irritating discharge, dirt and uncleanliness; some, but by no means all, are gonorrhœal. They grow very fast during pregnancy.

Cysts of Bartholin.—These are generally retention cysts of the duct; the gland is rarely affected; they are due to inflammatory occlusion of the orifice of the duct; gonorrhœa being a frequent cause of the occlusion. Such cysts are very common, and generally occupy the lower third of the labium majus, forming a bulging swelling, best seen on the mucous surface, which is pear-shaped, with the small end uppermost. As they increase in size they extend downwards into the labium; they are elastic, and their walls in some cases excessively thin. They generally contain watery or viscid mucus, which is occasionally coloured from altered blood; sometimes the cysts may contain a material like putty or porridge. They are liable to inflame, and the cyst is then converted into an abscess. If the cyst arise in the gland itself the swelling is rounded and more deeply seated.

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[1] Herman, *Diseases of Women*, p. 492.

Abscess of Bartholin. This may occur either in the duct or gland; if in the **duct**, it is small and generally bilateral, and the abscess points where the duct opens, *i.e.* on the inner surface of the labium minus, in front of the hymen, low down. Normally the duct of Bartholin is half an inch long.

If the abscess occur in the **gland**, it is one-sided and forms a swelling which is deeply situated and which involves the lower portion of the labium, extending backwards towards the perineum. There is generally much induration and thickening, and the swelling is not so superficial as a cyst of the duct which has suppurated.

In some cases there is little swelling, but only induration, and no obstruction to the duct; yet pressure over the situation of the gland will cause pus to escape from the duct. Cases of abscess are frequent in gonorrhœa. If an abscess of the duct be cut into, its walls are very thick and exhibit an irregular septate cavity unlike an ordinary suppurating cyst.

Herman[1] says that abscess of the gland differs from abscess of the duct in being more deeply situated, slower in formation, larger, and more painful; it seldom discharges by its duct, and if it bursts the opening is not large, and the abscess wall is rough and thick, not smooth and regularly ovoid.

Bartholin's gland is situated above the deep perineal fascia below and outside the vagina, beneath the lower part of the labium majus; and is about an inch from the ischium, lying in the fat of the ischio-rectal fossa. It secretes the colourless, tenacious, mucoid fluid which lubricates the vulva.

Other kinds of suppurations of the vulva are seen in suppuration and breaking down of a hæmatoma due to traumatism or bursting of a varix, or to rectal or ischio-rectal suppuration, cellulitic abscess, or even boils.

Varices.—These are common in pregnant women and multiparæ, the colour and feel of the enlarged veins being typical; they may be symmetrical and involve both labia, or occur in more or less isolated patches. Sometimes the whole vulval and vaginal orifice is intensely livid and vascular, and the rectum, vagina, and urethra share in the varicose condition. The danger of varices is rupture, either by accident (sometimes with rapidly fatal hæmorrhage), or in labour as the child's head passes the vulval orifice pressing on the varix. This may be followed by the formation of a **Hæmatoma of the Vulva**, which is a serious condition; but it may occur in the absence of varicosity from mere increased tension and pressure; the blood so effused is limited by the planes of fascia in the neighbourhood. If the blood[1] be effused into the labia, it may travel beneath the superficial fascia up on to the abdominal wall; if effused above the level of the visceral layer of the pelvic fascia, it will not descend into the ischio-rectal fossa. The commoner site is below the pelvic fascia, and it may be very extensive, forming an elastic tense swelling of a dark purple colour on one side of the vulva, associated with great pain. The tension may check the bleeding, or the blood may travel for an indefinite distance in the abdominal wall; or the skin and tissues may give way and the blood escape at the surface; the wound may then suppurate and grave septic symptoms arise.

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[1] Herman, *Diseases of Women*, p. 506.

Enlarged veins about the vulva and abdominal wall also occur with abdominal tumours; they indicate pressure or obstruction to the pelvic venous circulation. Tumours giving rise to enlarged veins over the abdominal walls are often malignant.

Hernia into the Labium descends in, or by the side of, the processus vaginalis of Nuck, entering the labium by the inguinal canal. It is less common than femoral hernia in women.

Another variety of hernia is described by Smyly,[2] in which the bowel descends either in front of, or behind, the broad ligament, passing down by the side of the vagina into the labium. Such a hernia may be called '**Perineal.**' Two different kinds occur:

In the first variety the hernia descends **in front of the broad ligament** distending the vesico-uterine fold of peritoneum, and passes down between the bladder and uterus, along the side of the vagina, into the labium.

In the second, it descends **behind the broad ligament** between the rectum and vagina either into the labium or perineum. Such a hernia may contain the uterus and ovaries, as well as intestine and omentum. These herniæ are liable to be mistaken for cysts of Bartholin's gland. They are very rare.

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[1] Dakin, *Handbook of Midwifery* (Appendix), p. 587.

[2] Smyly, *op. cit.* Allbutt and Playfair, *System of Gynæcology*, p. 380.

Fibromata of the Vulva generally occur in the labia majora and may attain a considerable size. They may also grow from the perineum or nymphæ. They may have a long stalk, and are sometimes flabby and soft from mucoid degeneration. 'Molluscum Fibrosum' is a name sometimes applied to these growths; they are liable to be mistaken for cysts of the labium.

Lipomata generally grow from the labia majora or mons Veneris; very large tumours are on record. They may resemble cysts of the labia, or elephantiasis. They sometimes have connections with the fat of the ischio-rectal fossa, or they may distend the inguinal canal like a hernia.

Encysted Hydrocele of the Canal of Nuck occurs, coming down the inguinal canal, the processus vaginalis being distended at one part to form a cystic tumour, or there may be several constrictions occupying the canal like a string of beads. Sometimes the hydrocele is of the congenital variety, being reducible, and having a well marked impulse. Hydroceles generally occupy the upper and middle part of the labium, the lower end being free.

Rupture of the Female Perineum.[1]—This subject belongs rather to midwifery, but its secondary results are seen frequently in gynæcological practice.

Rupture is almost invariably caused by parturition. It may be due to malpresentations, especially unreduced occipito-posterior positions of the vertex, the use of instruments, to want of care in delivery of the head, or more frequently the shoulders, to very rapid labour, and to a large child. Primiparæ, especially old primparæ, are liable to rupture.

The perineum is rarely in itself diseased. Sometimes a perineum is very soft, at other times rigid or inelastic from chronic inflammation, but this is rare. A perineum that has been previously repaired may of course be inelastic in a subsequent labour. Strong uterine action, and sudden stretching, are important factors. The amount of tearing also depends upon the length and situation of the perineum. The length of the perineum in a multipara varies from five-eighths of an inch to two inches; the situation of the fourchette varies from as much as two inches behind the lower border of the symphysis to close up to it. If the perineum be long and its anterior edge be far forward, rupture is more liable to take place.[1]

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[1] Matthews Duncan, *Papers on the Female Perineum, and on the Methods proposed to prevent Rupture.* Also Berry Hart, *Atlas of Female Pelvic Anatomy,* Edin. 1884; Herman, 'A Contribution to the Anatomy of the Pelvic Floor,' *Trans. Obstet. Soc. Lond.* vol. xxxi. 1889, p. 263; and *id.* 'On the Changes in the Pelvic Floor which accompany the slighter degrees of Prolapse,' *ibid.* p. 276.

The tension in labour being from side to side, the line of tearing is always in a line perpendicular to that of greatest tension, *i.e.* backwards, and generally in the middle line. The fourchette is always torn with first labours; and in sixty per cent. of cases in first labours the tear extends further and involves the skin of the perineum (M. Duncan). If the sphincter be not involved, such a tear is called **incomplete**; if the sphincter is torn, then it is **complete**. The tear in the posterior vaginal wall and rectum generally passes to one or other side of the posterior vaginal column.

Central Rupture is very rare. The tear is situated between the anus and fourchette, the anterior portion of the perineum being intact. It is caused by the head forcing aside the muscular structures of the perineum, bulging the skin, which finally gives way.

Rarely the skin of the perineum may split, and yet the mucous membrane and cellular tissues are intact[2]; exactly the reverse may also occur. Very rare cases are reported where the child has been born through the posterior strait of the pelvis, tearing the recto-vaginal septum, being finally born through the anus, with laceration of the perineum forwards.

The chief results of rupture of the perineum are that the raw edges form a surface for septic absorption, and should for that reason be at once closed; and even if repaired some days after, if the granulation tissue be thoroughly removed and the edges refreshed and sutured, the case will do well.

Another serious result of complete rupture is incontinence of fæces, or at all events of liquid fæces and flatus. These cases, if left alone, may recover to a certain extent, but generally need a planned operation for repair later on.

Neither complete nor incomplete rupture causes descent of the womb. The perineum does not support the uterus, though a ruptured perineum does away with the support that a pessary requires in the treatment of cases of prolapse ; therefore a perineum is repaired in order that a patient may wear an instrument.

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[1] Herman, 'Injuries in Parturition,' Allbutt and Playfair, *op. cit.* p. 433.

[2] Herman, *op. cit.* p. 435.

If the perineum be not repaired the surface gradually becomes covered with granulation tissue, with a more or less variable amount of scar tissue, and if **incomplete** the vulval outlet may appear to be prolonged backwards, and on separating the labia a shining surface of cicatricial tissue on the posterior vaginal wall marks the amount of laceration ; the sites of the torn ends of the transversus perinei and bulbo-cavernosus cannot, of course, be detected. In chronic cases there is often both cystocele and rectocele on straining. The levator ani and sphincter are intact, and the sling-like fibres of the former surrounding the vagina can be easily felt.

If the rupture be **complete** (after the parts have cicatrised) the anus is represented by an opening of a triangular shape, its base posteriorly formed by the anus, being corrugated and concave, the sides of the triangle being formed by the edges of the torn recto-vaginal septum. The apex of the rupture points upwards, generally more or less to one side. The tear may extend some distance up the vagina. The sphincter ani is completely torn through ; the little furrows existing around the anus (caused by the corrugator cutis ani) are only present posteriorly ; the ends of the sphincter are retracted, there is no grip to the finger introduced into the bowel, the bright red mucous membrane of which can easily be seen. There is often some prolapse of the bowel as well.

The contraction of the scar tissue in healing sometimes produces very great distortion of the parts, and gives trouble in the later operations for repair.

CHAPTER II

ON DISEASES OF THE VAGINA

Age Changes.—The vagina in the child is a mere transverse slit; the folds and rugæ are, however, well marked, and the walls are in close apposition.

At puberty the vagina becomes larger, especially at its upper part, the folds are well marked, and the walls assume a bright pink colour. With marriage and childbirth the vagina is stretched, and its folds and rugæ more or less flattened, the orifice is widened, and the walls, both anterior and posterior, tend to protrude (cystocele and rectocele) (see Prolapse).

In many cases, in women who have had large families, and where repeated stretching has taken place and the perineum is deficient, the vulval and vaginal orifice is wide and gaping; it may admit even the closed fist, and no pessary can be retained; and instead of the walls being in contact as they should be, air enters, and the whole vagina is 'ballooned,' the rectum often sharing the same atonic condition.

The expulsion of air from the vagina in such cases,[1] sometimes with noise, as the patient moves or sits down, may become very distressing (vaginal flatus).

The same ballooning of the vagina, and the entrance of air, occurs when a patient is placed in the knee elbow position, as in the reposition of retroverted gravid uterus.

At the menopause atrophic changes occur, the walls of the vagina become smooth and pale, and the cavity smaller, especially at the upper part, which is converted into a sort of cone, into the apex of which the atrophied cervix projects, or there may be no cervix, but only the dimple where the cervical canal opens, often difficult to feel.

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[1] Matthews Duncan, *Clin. Lect.* p. 177, 4th ed., 'Garrulitas Vulvæ.'

MALFORMATIONS OF THE VAGINA

These may be:

1. Double or septate vagina.
2. Congenital absence or defect.
3. Atresia, congenital or acquired.

Malformations of the vagina are frequently combined with abnormalities of the uterus (the chapter on this subject should be consulted), for both vagina and uterus are derived from Müllerian ducts.

Double or Septate Vagina.—In this case the two Müllerian ducts remain separate either in part or their whole extent, the vulva being single. A double vulva is extremely rare; Ballantyne [1] reports such a case, and places the individual among the double terata. In the ordinary double vagina with the single vulva the two canals are situated side by side; it is very rare for them to be situated antero-posteriorly.

Sometimes the canals are not exactly parallel, and one is often a little larger than the other. The septum may be complete or not, or there may be merely a ridge to mark the double condition. The uterus is generally double or septate, and with a double cervix. There may be atresia of one or both vaginal canals, giving rise to retention of menstrual blood.[2]

If pregnancy and labour ensue, the septum may possibly give trouble; it may be torn, sometimes with serious consequences, but in other cases is simply pushed aside. Dyspareunia may occur. The condition is sometimes only discovered accidentally. It has been mistaken for a vaginal cyst, where one canal is imperforate.

Unilateral vagina with uterus unicornis may occur.

Rudimentary or Defective Vagina.—Complete absence of the vagina [3] is a rare condition. Generally in the condition described as absent vagina some muscular bands still exist between the bladder and rectum. This should really be described as atresia [4] of the vagina, or imperforate vagina. In true absence of the vagina there is nearly always absence or

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[1] Ballantyne, Allbutt and Playfair, *op. cit.* p. 85.

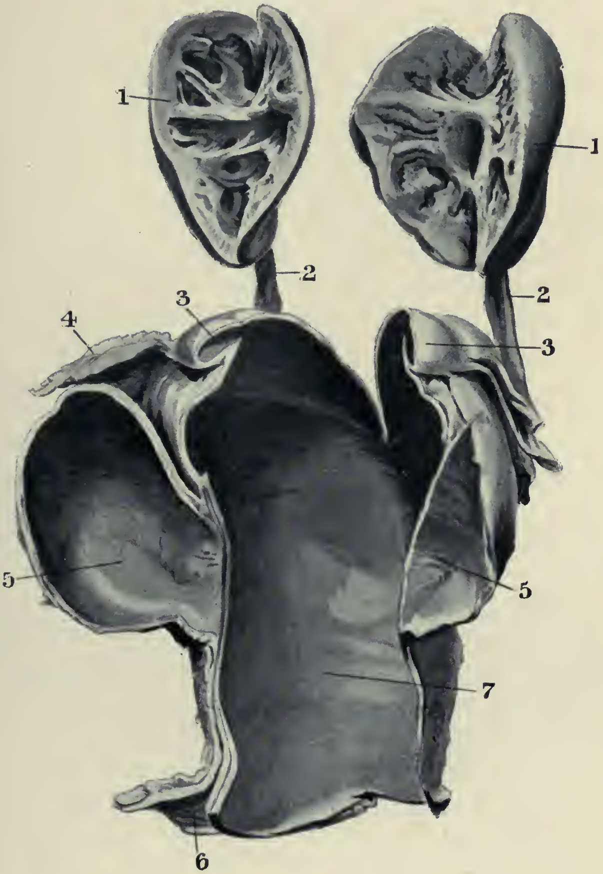
[2] See chapter on 'Disorders of Menstruation.'

[3] Lewers, 'A Case of Double Uterus with Double Hæmatometra and Complete Absence of the Vagina,' *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 327.

[4] Ballantyne, *op. cit.* p. 87.

FIG. 4.

IMPERFORATE VAGINA IN AN INFANT, TO SHOW RESULTS OF ATRESIA.



The urino-generative organs of an infant 7 weeks old. The vagina is seen as a large sac, which was distended with puriform fluid owing to vaginal atresia at the lower extremity. The bladder is enlarged and was drawn up out of the pelvis. There is also great distension of the ureters and kidneys, owing to the pressure of the distended vagina. The uterus is seen in section on the summit of the tumour, almost unaltered. The specimen resembles hæmatocolpos in the adult.

1, 1. Dilated kidneys.
2, 2. Dilated ureters.
3, 3. Uterus in section, on the summit of the distended vagina.
4. Fallopian tube.
5, 5. Bladder.
6. Urethra, much elongated.
7. Large sac formed by the greatly distended vagina, which was imperforate at its lower extremity.

(Specimen 3016 A. Museum, St. Bartholomew's Hospital, Drawings, Series 57, Nos. 972, 973.)

rudimentary development of uterus, tubes, and ovaries. The external genitals and breasts are also ill developed. Clinically, this condition cannot be distinguished from atresia of the vagina.

Vaginal Atresia or Imperforate Vagina.—The results of this condition are treated of under Retention of Menses.

In its most marked form no trace of a vagina exists except some fibrous or muscular tissue existing as a band between the bladder and rectum, or, falling short of this, the vagina may be partially perforate, the rest being solid. In a less marked form[1] there is simply a membranous obstruction or perforated diaphragm at one part of the passage. Sometimes the lower part of the vagina is patent, sometimes the upper, the remainder being imperforate.

Ballantyne points out that when the upper two thirds of the vagina are occluded, it has been supposed that the open lower third is not truly vaginal in nature, but is the enlarged vestibular canal, the representative of the anterior part of the sinus urogenitalis of intra-uterine life. When only the middle part of the vagina is obstructed, it may be surmised that the upper canal is Müllerian or truly vaginal in character, whilst the lower is vestibular.

Great differences exist in the other genital organs with vaginal atresia; the uterus may be normal or rudimentary, the vulva more or less imperfect, though generally it is normal as well as the hymen. The ovaries are generally present; sometimes the urethra has been widely dilated by coitus.

The conditions are generally not noticed till the onset of puberty, when the menstrual products are retained.

It is very rarely that vaginal atresia gives rise to any trouble in infancy, but a curious specimen[2] exists in St. Bartholomew's Hospital Museum (Fig. 4), where muco-pus accumulated in the vagina of an infant with this condition, producing similar symptoms to hæmatocolpos in an adult. The vagina was enormously distended. The condition was not diagnosed during life. The sac caused great distension of the uterus and kidneys from pressure.

Acquired Atresias are generally of traumatic origin, or due to puerperal ulceration and sloughing with subsequent contraction of scar tissue.[1]

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[1] Ballantyne, *op. cit.* p. 87.

[2] St. Bartholomew's Hospital Museum, No. 3016A. *Ibid.* Drawings 972, 973.

Abnormal Communications of the Vagina.—The vagina may open into the rectum when the recto-vaginal septum is imperfectly developed; or into the urethra or bladder, resembling the cloacal conditions of embryonic life. Ballantyne[2] classes them as really vulvar anomalies. Cases of congenital ano-vaginal and vagino-urethral fistulæ are very rare.

THE QUESTION OF VAGINAL SECRETION AND VAGINAL GLANDS

There are differences of opinion on the point; in health the vagina is certainly moist, and the secretion is thin, whitish, opalescent, and opaque, like thin starch, slight in amount, and in the vagina has a strong acid reaction.

Great care must be taken in such cases to eliminate discharges which may come from above, and, as such, do not belong to the vagina at all, and to take into account the possible changes which may occur in a secretion which is retained and therefore subject to the influence of micro-organisms from without.

Gow[3] investigated the subject, and points out that the secretions *found* in the vagina are derived partly from the uterus and partly from the vagina itself; but in order to eliminate the uterus, the secretion was investigated in cases where the uterus had been removed for malignant disease, and where the vagina was healthy. Gow found that the whole vagina still remained moist, and the secretion acid, so the assumption was that the vagina did secrete.

As to the question of **glands** in the vagina, certainly their number is exceedingly limited, but we may have secretion without glands as such, since glands are but involutions of a surface which secretes, in order to increase that surface. The rugæ of the vagina may act in the same way by increasing the surface by elevation instead of depression. The surface of the vagina is very like skin, and there is desquamation of the superficial layers, with a certain amount of active transudation of albuminous fluid, as in sebaceous glands. In the vaginal secretion, flattened nucleated cells appear similar to those covering the vagina; the secretion, mixed with water and boiled, coagulates, containing a proteid material belonging to the albumins or globulins.

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[1] Puech, 'Complex Atresias of the Female Genital Passages,' *Ann. de Gynécologie*, Paris, 1895.

[2] Ballantyne, *op. cit.* p. 91, and literature.

[3] 'A Note on Vaginal Secretions,' *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 52.

Very little, if any, mucin is present.

If the secretion of the vagina be observed *in situ* it is acid, but if all air and micro-organisms are excluded *as secreted* it is alkaline, and the subsequent acidity is due to decomposition of the material secreted. Both Gow and Döderlein[1] come to the conclusion that micro-organisms exist in the vagina. The vaginal bacillus, a rod-shaped organism belonging to the anaërobic bacilli, can be cultivated in bouillon containing one per cent. of sugar, developing a free acid (lactic),[2] and it is probable that the acidity of the vaginal secretion is due to this, or other micro-organisms, but that if such are carefully excluded the reaction of the secretion is alkaline. Whether such micro-organisms[3] found in the vagina are pathological or not is a matter of debate, but the anatomical structure of the vagina is said to preserve it against invasion even of saprophytes, the tubercle bacillus, or the gonococcus, unless the surface be previously injured.

Döderlein[4] says further that in new-born children the reaction of the vaginal secretion is neutral; in virgins and in normal pregnancy the acidity is greater, whilst in pathological conditions the reaction is feebly acid, neutral, or even alkaline; the acidity disappears during and for some days after menstruation and for five or six weeks after normal labour, and that, in the presence of the vaginal bacillus and its acid reaction, saprophytes and pathogenic micrococci are unable to develop and before long perish, and therefore that the vaginal bacillus has a protective influence against infection; and further that when pathogenic organisms are found in normal vaginal secretion they are always in a condition of weakened virulence.[5]

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[1] Döderlein, *Das Scheidensekret*, Leipzig, 1892.

[2] Butyric (Gow).

[3] Vaginal bacillus, Monilia candida, etc. See chapter on Micro-organisms in relation to Diseases of Women in this book.

[4] Bland-Sutton and Giles, *Diseases of Women*, p. 112.

[5] Winter, quoted by Bland-Sutton and Giles.

Probably some vaginal cysts[1] contain fluid representing the normal vaginal secretion.

Pathological Discharge from the Vagina.—This is important, but many factors modify its characters. It may be a symptom of vaginitis, and as such, yellow, greenish, or even purulent, and mixed with viscid mucus. It is altered by the discharges of menstruation, childbed, uterine catarrh, cancer, sloughing fibroids, irritating urine or fæces in fistula, or decomposing secretions in foreign bodies. Injuries or irritating douches may inflame or irritate the vaginal walls. Gonorrhœa of course is to be thought of, but the vagina is less seldom affected than the vulva and cervix, while certain diseases, such as tuberculosis and the exanthemata, may play a part. Nothing, perhaps, is more difficult than to determine whether a discharge is to be described as pathological or not, and in connection with the vagina its exact source must be discovered if possible; often not an easy matter. If a normal discharge be in excess it may border on the pathological, while purulent, watery, fœtid, or blood-stained discharges certainly are so.

Vaginitis and its Causes.—It is very difficult to make a reasonable classification of the varieties and causes of vaginitis: some authors attempt such from the standpoint of its causation; others, from the appearance presented, or according to the age at which the disease occurs. Some cases of vaginitis are primary, others again are secondary, depending upon irritating discharges from above, or infection from below; the whole subject is confusing.

Vaginitis or Colpitis may be due to :—

a. *Injuries*, as in obstetric operations, accidents, instrumentation, foreign bodies,[3] and the like.

b. *Infections*, particularly in connection with pregnancy and parturition; gonorrhœa and tuberculosis are less common causes.

Clinically, there may be[4] :—

a. The vulvo-vaginitis of children.

b. Vaginitis of pregnant and puerperal women.

c. Gonorrhœal vaginitis of adults.

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[1] Rutherfoord, *Trans. Obstet. Soc. Lond.* vol. xxxiii. 1891, p. 354.

[2] Bumm, *Veit's Handbuch*, vol. i. p. 474.

[3] See Bland-Sutton and Giles, *Diseases of Women*, 1st ed. p. 108.

[4] Bland-Sutton and Giles, p. 119.

d. Senile vaginitis.

e. The so-called membranous and diphtheritic vaginitis, associated with the exanthemata or dysentery.

Or, from the *frequency of occurrence*, vaginitis may be divided into :—

1. Common forms.—Catarrh (simple, puerperal, or gonorrhœal); vaginitis caused by irritating discharges or foreign bodies; injury, and bacterial infection; senile vaginitis.

2. Rare forms.—Purulent vaginitis; granular vaginitis; pustular vaginitis; vaginitis with gas cysts; membranous vaginitis; erysipelatous vaginitis.

Furthermore, vaginitis may be **acute** and **chronic**; still the writer finds himself, beyond mentioning the above facts, unable to put the matter in any clearer light, and hence only some of the varieties which seem to be of the greatest interest will be described. Herman,[1] perhaps more reasonably, treats of the whole subject under the symptomatic heading of 'Leucorrhœa' (*q.v.*).

Pathological Anatomy.—Vaginitis seems to present itself in many stages. There may be simple catarrh with increased discharge, or acute inflammation with profuse purulent discharge of pus or blood, or even necrosis, exfoliation, and gangrene.

In fairly acute cases, from whatever cause, there is increased vascularity and redness of the mucosa, the discharge is turbid or purulent, yellowish or greenish, as the case may be. The epithelium is swollen, due to an infiltration of round cells in the papillæ; the rugæ are exaggerated, swollen, bright red, and easily bleed, or they are obliterated by the processes of exudation and the epithelium desquamates and thins, showing the papillæ below, which gives the granular appearance to such cases. Large patches of epithelium may even slough and be detached, mixed with coagulated exudation, from the acuteness of the inflammatory process (exfoliative or membranous vaginitis); the exposed surfaces of the vagina subsequently cohere, and may give rise to serious atresia. In such cases the deeper parts of the vagina and cellular tissues are involved in a sort of phlegmonous inflammation of the severest type; they occur in puerperal cases, and with the exanthemata.

The following varieties of vaginitis will be shortly described :—

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[1] *Diseases of Women*, p. 424–473.

1. **Simple Catarrh.**—This is seen in children or young adults, and is of a simple nature. Leucorrhœa is the only symptom; it is unimportant.

2. **The Vulvo-vaginitis of Children.**—This is important, but the writer cannot believe that the majority of cases are gonorrhœal;[1] and, further, he believes that in infants the vulva is generally affected alone, vaginitis being rare. The question has legal importance, and much depends upon the certainty with which the gonococcus of Neisser can be identified, especially in the presence of other organisms; pus certainly proves nothing. It is even possible that the vulvo-vaginitis of children is due to a special micro-organism.[2]

For the results of Vaginitis in children in relation to Salpingitis, the reader is referred to Herman, 'Diseases of Women,' p. 428.

3. **Gonorrhœal Vaginitis of Adults**[3] possibly occurs, but it is much more common for the brunt of the disease to fall on the vulva, urethra, and cervix, and, later, on the tubes and peritoneum.[4]

If the vagina be affected, the acute stage only lasts a few days. The onset is sudden; there is swelling and thick yellow discharge; the pain is due to the inflammation of the vulva and urethra, not the vagina. The discharge diminishes in a few days, and may then become a chronic leucorrhœa, with no diagnostic points whatever. It would be very unwise to make the assertion that a case was gonorrhœal without fairly certain evidence elsewhere than in the vagina.

As before stated, the gonococcus,[5] if present, is valuable evidence, and the history may further help us, but histories are too frequently unreliable. The cultivation test of the gonococcus should, if possible, be applied.[6]

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[1] Drummond Robinson, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 14.

[2] McCann, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 225, 'Etiology of Gonorrhœa.'

[3] *Id.*, *op. cit.* and article 'Gonorrhœal Infection,' *Encyclopædia Med.* vol. iv. p. 191.

[4] Cullingworth, 'The Etiological Importance of Gonorrhœa in relation to some of the more common Diseases of Women,' *Brit. Med. Jour.* vol. ii. p. 122, July 20, 1889; also White, 'Oöphorectomy in Gonorrhœal Salpingitis,' *Brit. Med. Journ.*, vol. i. 1889, p. 285.

[5] Herman, on the 'Diagnostic Value of the Gonococcus,' *op. cit.* p. 436.

[6] See chapters on 'Gonorrhœal Inflammation in relation to Diseases of Women,' also chapter on 'Micro-organisms.'

4. **Puerperal Vaginitis.**—This may be severe, especially after a difficult delivery, and where lacerations have taken place. Its importance largely depends on the question of septic infection. The bruising and laceration may be serious, and sloughing from the severity of the injuries may lead to formation of fistulæ, or to serious contractions later. The subject belongs rather at this stage to midwifery than gynæcology, but foul discharges may exist in the vagina, and the uterus be quite free; hence the danger sometimes of douches after labour unless the vagina itself has been first properly washed out.

Later, puerperal vaginitis may be seen together with imperfect involution of the uterus; there is discharge and catarrh, and the tissues are swollen and easily bleed. The cervix is also often lacerated, everted, eroded, and inflamed. The trouble all dates from the confinement, not an uncommon thing in diseases of women.

5. **Senile Vaginitis.**—This is not an uncommon condition in old women, or women past the menopause; it is important, in that it is sometimes confounded with cancer of the cervix, and also in the liability of the disease to cause curious constrictions in the upper part of the vagina itself. The vagina in such cases feels like a cone very much narrowed above, at the apex of which the cervix, or the dimple formed by the os externum, can just be felt. Sometimes bands of contracting scar tissue can be felt encircling the vagina, or even a distinct diaphragm may exist through which the finger can be passed, and which divides the vagina into one or more loculi. The diaphragm is taken sometimes erroneously for the cervix, but if the finger be passed through it, the cervix is found above the constriction. The change affects the upper part of the vagina mostly. The vaginal walls are smooth and white in places; in others there are reddish spots and areas. There is denudation of epithelium, probably from defective nutrition, and granulations form. There may be a purulent reddish discharge, which, if retained in the pockets high up, decomposes, and is very offensive, simulating cancer. The scar tissue around the vaginal walls has a remarkable power of contracting, producing great diminution of the vaginal canal. The cause of the condition is doubtful; it may occur with foul pessaries. Herman[1] suggests gonorrhœa, or alcohol, or, in

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[1] *Diseases of Women*, p. 463.

some cases, a condition possibly allied to that described by Matthews Duncan as lupus (esthiomène). It is also associated with pruritus.

6. **Purulent Vaginitis** (not gonorrhœal).—This is described by Herman as a condition where the chief feature of the case is the abundant secretion of pus[1] for long periods, and the vagina is red but not granular; it is not gonorrhœal or due to an abscess discharging into the vagina.

7. **Granular Vaginitis.**—This may be a stage of chronic inflammation of the vagina, where, either from exposure or from the constant wearing of a pessary, the wall may become roughened, thick, and more or less shotty on examination; it also occurs in women with chronic discharges from the cervix. Here the word 'granular' is applied merely to describe the condition as felt clinically. It is common. Herman[2] and Deville describe a rare form of granular vaginitis in which there are distinct papules scattered over the whole vagina and on the cervix; they feel like shot embedded in the mucous membrane. The papules are red in colour, but they are probably not inflamed glands. It chiefly affects married women. It may be the result of gonorrhœa. It may date from the beginning of pregnancy. There may be a copious purulent discharge. Its cause is unknown.

8. **Pustular Vaginitis.**[3]—This is very rare; the condition is marked by the occurrence of pustules like those of smallpox in the vagina. One case occurred after pregnancy, and the patient had been possibly exposed to both gonorrhœa and syphilis. There was a very thick yellow discharge.

9. **Vaginitis with Gas Cysts.** (Emphysematous Vaginitis, or Colpo-hyperplasia cystica.)—This is a rare disease. Winckel has described cases in Germany, Gervis and Herman[4] in England. The disease resembles granular vaginitis, but the papules or pustules contain gas cysts.

These gas cysts look greyish or purple, and if punctured under water a bubble of gas floats up; after the vesicles are pricked they disappear. This disease may occur in pregnancy or apart from that condition. It may occur also with gonorrhœa.

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[1] Herman, *Diseases of Women*, p. 438.

[2] *Op. cit.*, p. 439.

[3] Herman, *Obstet. Journal*, vol. vii.; *Diseases of Women*, p. 440.

[4] Herman, *Lancet*, vol. i., 1891, p. 1252, with literature; *Diseases of Women*, p. 441.

Herman thinks that it is a stage of granular vaginitis. Haultain[1] classes it as inflammatory. Matthews Duncan[2] says it is probably not an inflammation. It affects the the higher part of the vagina, and the gas 'is said to be tri-methylamin.'

10. **Membranous Vaginitis.**—This is a rare condition, but in some cases shreds or actual casts of the vagina have been passed; it has occurred after strong caustics or douches. By some it is thought to be allied to exfoliative dermatitis. In some cases the exfoliation is said to be periodical, and associated with dysmenorrhœa.[3] Other cases of so-called membranous vaginitis are said to be due to fungi, such as Oïdium albicans, Monilia candida, and Leptothrix vaginalis (Colpitis mycotica).[4]

White membranes may form on torn vaginal or vulval surfaces as the result of acute puerperal infection.

The exanthemata, measles, smallpox, and typhus, also dysentery and diphtheria, are said at times to be attended with so-called membranous vaginitis. In some cases the cellular tissue is also affected (erysipelatous or phlegmonous peri-vaginitis); this is called 'para-colpitis dissecans' by Matthews Duncan.[5]

The question of the relation of vaginitis to constitutional conditions is discussed by Matthews Duncan; alcoholism, old age, lupus, and diabetes[6] being given as causes.

Vaginismus[7] is a term applied to an abnormal hyperæsthesia of the external genital organs causing muscular spasm, and occurs chiefly in young nervous hysterical women. Its chief phenomenon is spasm of the vaginal orifice, and this may be a cause of dyspareunia. The condition is difficult both to describe and investigate, and there appear to be a variety of cases. They fall roughly into two groups:[8]

1. Primary vaginismus, a neurosis.
2. Secondary vaginismus, where a definite local cause can be discovered.

Primary cases are those in which the vaginal spasm is functional; there is no structural change to be found on examination, and the patients themselves are the subjects of hysteria and the allied neuroses. The chief element of the condition is spasm of the levator ani, and in a well marked case the constriction caused by the fibres of this muscle as it surrounds the vagina can be well felt; the sphincter ani and other muscles of the perineum may share in the spasm, and the condition is sometimes associated with dysmenorrhœa. The spasm may be brought about by the slightest touch or even attempt at vaginal examination. Patients of this sort are often intensely neurotic, and so great is the agony complained of, coupled with evidence of extreme terror on any attempt at examination, that convulsive seizures, opisthotonos, or even syncope may occur.

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[1] Allbutt and Playfair, *op. cit.* p. 389.

[2] *Clin. Lect.* 4th ed. p. 181.

[3] Haultain, Allbutt and Playfair, *op. cit.* p. 389.

[4] Haultain, *op. cit.* p. 388.

[5] Matthews Duncan, *Clin. Lect.* 4th ed. p. 171.

[6] *Ibid.* p. 173.

[7] Marion Sims, *Trans. Obstet. Soc. Lond.* vol. iii. 1861; *Amer. Med. Times*, 1862.

[8] Champneys, *Clin. Journal*, Nov. 16, 1892.

Sometimes the condition is less severe, and is only brought about by any attempt at stretching the levator ani within the vagina.

In other cases the subject seems to be a sexual one, accompanied with frigidity or absence of the sexual desire, and only occurs on sexual approach, not necessarily on touch or examination. It may occur in young women who marry old decrepit husbands, where sexual affinity and compatibility do not exist. Should pregnancy occur it may persist even after parturition, and it may even obstruct labour.[1] It may be one-sided only, it may develop suddenly, or it may vary in its intensity: it has no regular tendency to spontaneous cure. It is a disease which is most frequently discovered on marriage, when sexual intercourse is found to be painful, difficult, or impossible.

Secondary Vaginismus.—This depends upon a local lesion [2] which can be discovered on careful local examination. A painful spot or fissure of the fourchette, vaginitis either acute or chronic, inflammation of the vestibule or hymen, caruncle of the urethra, or caruncular conditions of the vestibule with little red tender spots, vulvitis or urethritis, sometimes tender prolapsed ovaries, or even anal fissure, may be discovered, in fact many of the causes of dyspareunia; yet all cases of dyspareunia are not cases of vaginismus The prognosis [3] of the cure of secondary cases is good, but of the primary, uncertain and unsatisfactory.

Injuries of the Vagina.—These occur generally in delivery, or from the use of instruments. All sorts of foreign bodies [4] may be found in the vagina, and pessaries have been forgotten and caused sloughing and perforations.

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[1] Matthews Duncan, *Clin. Lect.* 4th ed. p. 159.

[2] Hart and Barbour, *Manual of Gynæcology*, 1886, p. 501.

[3] C. Lee, Mann's *Amer. Syst. of Gynæcology and Obstetrics*, vol. ii. p. 36.

[4] For a list of foreign bodies found in the vagina, see Bland-Sutton and Giles, *Diseases of Women*, 1st ed. p. 108.

The chief importance of injury to the vagina is the possible occurrence of **fistulæ.** They generally result from sloughing after protracted labour; tearing of the tissue or perforation by the use of instruments is rarely seen, and, if so, it is the recto-vaginal wall, not the vesico-vaginal, which is generally injured. Foreign bodies, as above mentioned, may cause fistulæ, as may also malignant disease of the cervix, while vesical calculi have ulcerated through from the bladder.

The chief varieties of such fistulæ are:—

1. Vesico-vaginal.
2. Recto-vaginal.
3. Urethro-vaginal.
4. Uretero-vaginal.
5. Vesico-cervical.
6. Uretero-cervical.

More will be said on this subject in the chapter on Diseases of the Bladder. Attention is generally directed to them by the distress they cause by incontinence of urine or fæces. Their detection is sometimes difficult, especially the utero-vesical and uretero-vaginal forms.

Vaginal Hernia.—This is a rare condition; it consists generally of a descent of Douglas's pouch, in which coils of intestine may sometimes be felt.

The part played by descent of the pelvic floor and descent of the uterus is discussed under Displacements of the Uterus, as are also descent of the anterior and posterior vaginal walls; descent of the pelvic floor is itself a hernia.

Vaginal Cysts.[1]—Though rare, these are of some importance. They generally occur (66 per cent.) in the lower part of the vagina, and on the anterior wall. As a rule, small cysts give rise to no trouble and are only accidentally discovered. Large cysts resemble prolapsed vaginal walls, for which patients mistake them. They cause trouble sometimes by bladder or urethral pressure, or they may inflame and suppurate. Vaginal cysts vary much in size, and are generally more or less rounded in shape, sometimes elongated in the long axis of the vagina, and are more or less sacculated or multiple; the largest cyst on record equalled a fœtal head.[1] They are rarely larger than an orange. They may become pedunculated; if so, they generally grow from the posterior wall of the vagina. The cyst wall varies in thickness, in some cases appearing translucent and thin; in others the wall is very thick. The cyst itself may be superficial or deep.

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[1] Rutherfoord, 'Cysts of the Vagina, their Etiology, Pathology, and Treatment,' *Trans. Obstet. Soc. Lond.* vol. xxxiii. 1891, p. 354; and A. Routh, 'On Cases of Associated Parovarian and Vaginal Cysts from a Distended Gartner's Duct,' *ibid.* vol. xxxvi. 1894, p. 152.

The fluid is generally clear, limpid or yellowish; in other cases viscid, mucoid, or tarry.

Vaginal cysts probably grow slowly, they generally occur in married women of middle age, but no period of life is exempt. They have been seen in children. They may discharge their contents into the vagina or urethra.

The cyst is covered with the squamous epithelium of the vagina; the wall consists of connective tissue and muscular elements, and the lining membrane is generally a low form of cylindrical cells without cilia, generally in a single layer, but there may be a stratified or pavement epithelium, or it may resemble endothelium.

Etiology.—The question is disputed, and much hangs upon the question of the existence of vaginal glands or crypts which serve as such, and resemble glands; other authorities believe them to be developmental in origin. Rutherfoord classifies vaginal cysts as follows:—

1. *Accidental.*
    - *a.* Originating in crypts of the vagina in a few instances by occlusion of their orifices and subsequent dilatation.
    - *b.* As effusions of serous fluid or blood into the connective tissue, generally during pregnancy and labour.
    - *c.* Arising from dilated lymph channels.
    - *d.* Arising in glands or diverticula of the urethra when occurring low down on anterior wall.
2. *Congenital.*
    - *a.* From persistent remains of Gartner's canals (Wolffian relics), which seems the most probable explanation. Some vaginal cysts situated high up may even be prolonged into the base of the broad ligament The Gartnerian (parovarian) duct can

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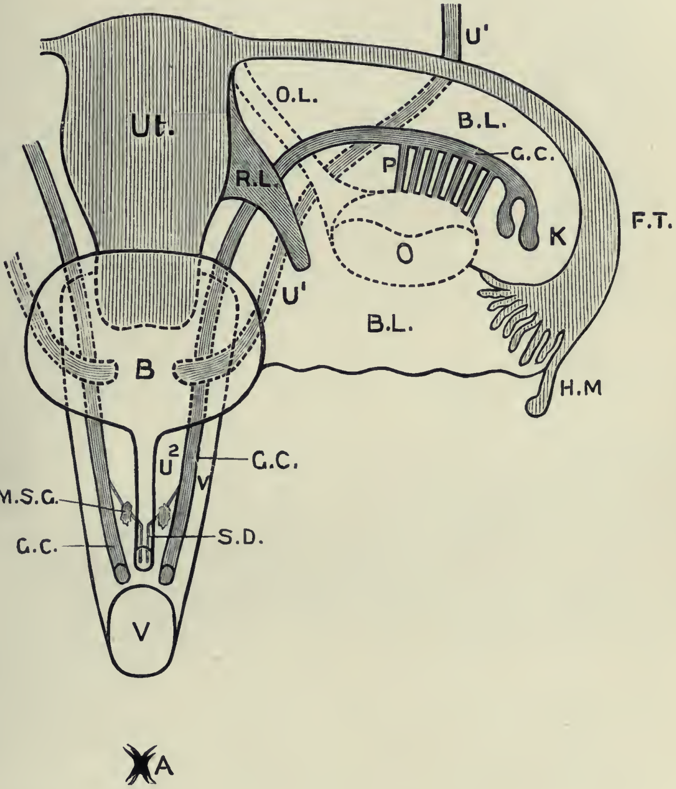
[1] Herman, *Diseases of Women*, p. 171.

Fig. 5.

DIAGRAM REPRESENTING THE FŒTAL STRUCTURES IN THE BROAD LIGAMENT AND VAGINA IN RELATION TO THE ORIGIN OF PAROVARIAN AND VAGINAL CYSTS. (Modified from Skene.)



Ut.	Uterus.
B.	Bladder.
U^1.	Ureter.
U^2.	Urethra.
S.D.	Skene's ducts.
V.	Vagina.
G.C.	Gartner's canals.
M.S.G.	Max Schüller's glands.
K.	Kobelt's tubules.
A.	Anus.
B.L.	Broad ligament.
O.	Ovary.
O.L.	Ovarian ligament.
R.L.	Round ligament.
F.T.	Fallopian tube.
H.M.	Hydatid of Morgagni.
P.	Vertical tubes of parovarium.

(Amand Routh, 'Trans. Obstet. Soc. Lond.' vol. xxxvi. 1894, p. 161.)

be actually recognised in some animals (cows and pigs) and traced to the vagina giving rise to cysts in those animals. In the human female it has been traced to the uterus, but it is doubtful if it has been satisfactorily traced further.[1]

Routh[2] (fig. 5) pointed out the association of Parovarian and Vaginal cysts, and explained their joint origin from a distended Gartner's duct, also how the fluid of vaginal cysts resembled parovarian fluid; further, how some cases of profuse watery discharges from the vagina of obscure origin might be accounted for by patency of Gartner's duct.

b. Arising from the Müllerian ducts, thick-walled cysts, which are deep-seated and lined with cylindrical epithelium.

Skene's ducts are probably quite distinct and belong to Max Schüller's urethral glands lying in front altogether of Gartner's ducts.

The developmental theory of vaginal cysts fails, however, to explain those occurring on the posterior vaginal wall.

Fibroids of the Vagina.[3]—These are very rare. They appear to be fibro-myomata and are always single, occurring in the anterior wall,[4] generally in the middle line, anywhere from the urethral orifice to the vaginal fornix. They are sessile as a rule, but may rarely become pedunculated.

They occur generally in the later sexual period or after the menopause. Only a very few cases are recorded in younger women or infants.

Most are small tumours the size of a walnut, though Phillips records one weighing over ten pounds, and Simpson[5] one as big as two fists. Herman, quoting Pozzi, speaks of one that filled the pelvis, and reached within a hand's breath of the umbilicus.

Pathological Changes.—Vaginal fibroids may undergo similar changes to those developed in the uterus, such as œdema, ulceration, calcification, and gangrene; cystic changes are also described. They may cause prolapse of the vagina.[1] As a rule the rate of growth is slow, but they may cause serious pressure symptoms, or may lead to sloughing of the bladder. Small fibroids give rise to no symptoms; they have no effect on menstruation, conception, or the course of pregnancy, but they may obstruct labour.

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[1] Gartner's duct is said to end in the vestibule just beneath, and to one side of, the urethral orifice.

[2] A. Routh, *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 152.

[3] John Phillips, 'Fibroids of the Vagina,' *Brit. Med. Journal*, vol. i. 1899, p. 262.

[4] Veit, *Handbuch*, vol. i. p. 348; Targett, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 101.

[5] Simpson, *Contributions to Obstetrics and Gynæcology*, p. 201.

Their origin is still a matter of dispute. Kiwisch holds that they arise in the uterus as cervical fibroids subsequently losing their uterine connection. Kleinwächter denies this.

Others hold that they arise in the vaginal or urethral wall itself, or in the retroperitoneal connective tissue. Fibroids may certainly arise from the retro-peritoneal tissue in the abdomen,[2] sometimes having wide bases and far-reaching connections.

Cancer of the Vagina (fig. 6).—This, as a *primary* disease, is rare. The chief forms are:—

a. Papillary squamous-celled epithelioma.

b. Infiltrating carcinoma, involving the whole vagina, generally seen in old women.

c. Sarcoma.
  1. Diffuse.
  2. Circumscribed (fibro-sarcoma).

Secondarily, the vagina may be involved in cancer of the cervix uteri, urethra, or vulva.

Secondary deposits occur in connection with sarcoma of the body of the uterus (deciduoma). The writer saw one case of this nature at the Samaritan Hospital under the care of Boulton, in which large oval nodules of a dark reddish blue colour rapidly appeared in the vaginal walls; the patient died from general metastasis. The same may occur in melanotic sarcoma.

Primary Epithelioma is a rare disease.[3] It is met with mostly in middle life; it generally commences as a raised irregular patch on the posterior vaginal wall; superficial ulceration[4] takes place, and the ulcer gradually spreads in all directions, and may perforate the recto-vaginal septum.

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[1] Specimen Lond. Hosp. Museum 2130. See drawing, Herman, *Diseases of Women*, p. 178.

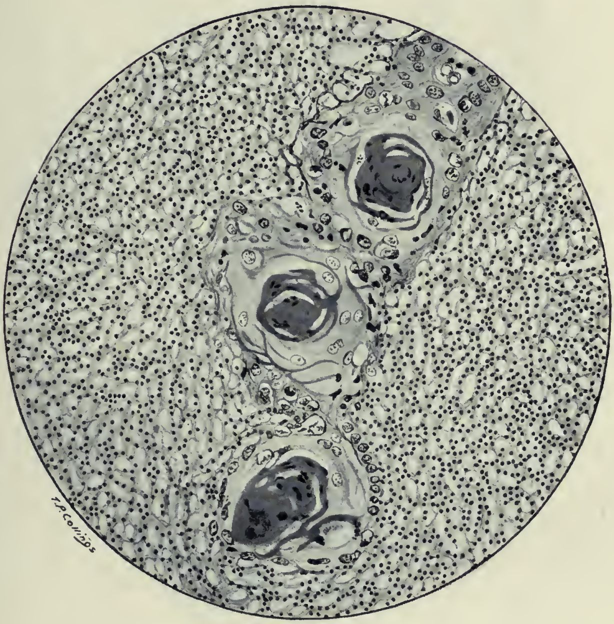
[2] Bland-Sutton, 'Tumour of the Mesometrium weighing 22 Pounds.' *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 298; Hecht, *Inaug. Dis.* München, 1891.

[3] Roberts, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 381.

[4] Küstner, *Arch. f. Gyn.* 1876, vol. ix. p. 279.

Fig. 6.

PRIMARY EPITHELIOMA OF THE VAGINA.



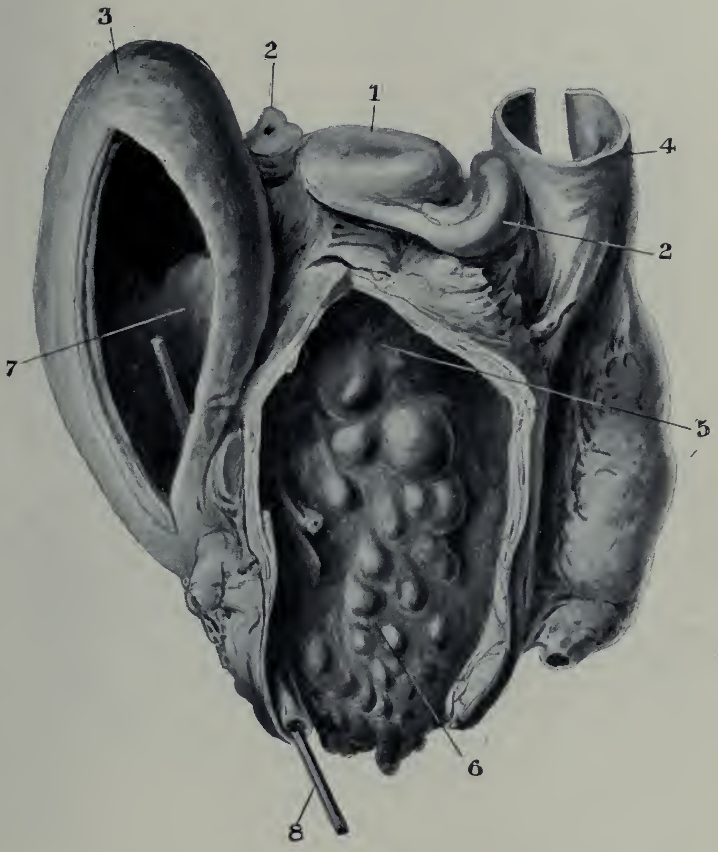
Section of a cancerous tumour which was removed from the posterior vaginal wall of a single woman of 38.

The growth is a very typical squamous-celled epithelioma with a few cell-nests. There is also much inflammatory infiltration. Three typical cell-nests are shown in the above drawing. (High power.)

(Section by H. J. Targett. See also Roberts, 'Trans. Obstet. Soc. Lond.' vol. xxxvii. 1895, p. 381.)

Fig. 7.

PRIMARY SARCOMA OF THE VAGINA. FIRST VIEW.



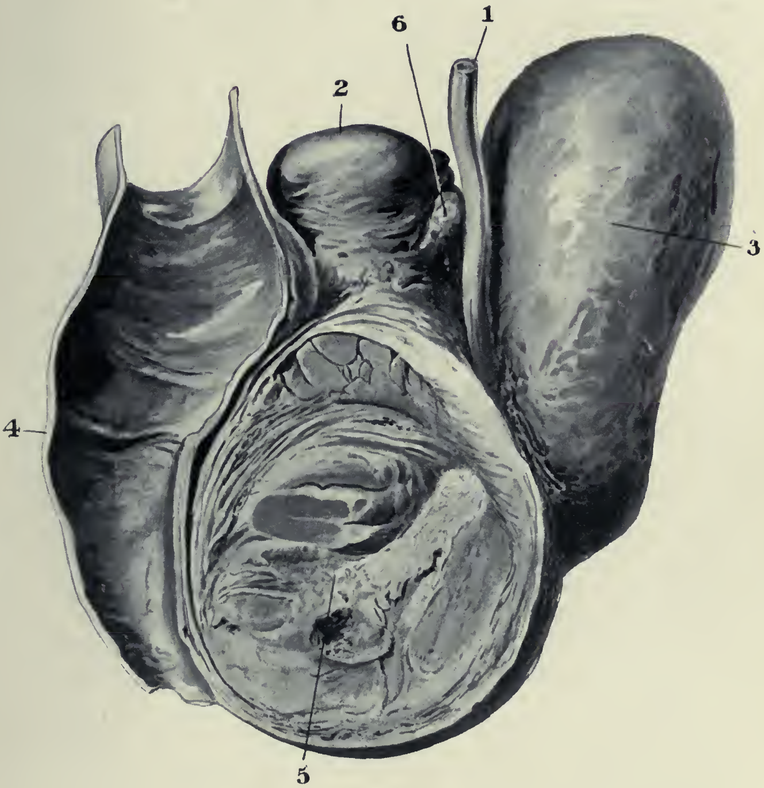
The pelvic organs of a child aged 2 years and 4 months, showing primary sarcoma of the vagina. The vagina is exposed by removal of its left wall.

1. Fundus uteri.
2, 2. Fallopian tubes.
3. Dilated bladder.
4. Rectum.
5. Portions of new growth in the vagina projecting into its cavity.
6. Polypoid masses of sarcoma in lower portion of vagina.
7. Portion of a sarcomatous mass protruding into the bladder.
8. Glass rod placed in the urethra.

(Specimen 3030 A. Museum, St. Bartholomew's Hospital. D'Arcy Power, 'St. Bartholomew's Hospital Reports,' 1895, p. 121, with references to published cases; *id.* 'Trans. Path. Soc. Lond.' vol. xlvii. p. 169.)

Fig. 8.

PRIMARY SARCOMA OF THE VAGINA. SECOND VIEW.



Section of the pelvic organs from the same case as fig. 7, showing a mass of new growth, which occupies the space between the rectum and bladder, and which bulges into the vagina.

1. The right ureter embedded between the vaginal wall and the bladder.
2. Fundus uteri.
3. Bladder.
4. The rectum.
5. A large mass of sarcomatous growth between the rectum and the bladder, which bulges into the upper part of the vagina (see fig. 7, in region marked 5).
6. Ovary and tube, of right side.

(Specimen 3030 A. Museum, St. Bartholomew's Hospital.)

Olshausen reports 18 cases, and of these 13 involved the posterior vaginal wall.

Primary Carcinoma sometimes occurs as a general annular infiltration and ulceration of the whole tube of the vagina, which becomes rigid and contracted.

Primary Sarcoma is remarkable for its occurrence, not only in adults, but in very young children [1] (figs. 7 and 8). The growth may be sessile, but is more frequently polypoid, forming masses like grapes, which protrude from the vagina; death occurs from wasting or interference with the bladder or rectum. It rapidly spreads to the external genitals and perineum.

In adults, sarcoma is usually of the diffuse form, and occurs as the round or mixed-celled variety, and there is a great tendency to necrosis of the growth.[2] If circumscribed, the growth is usually a fibro-sarcoma. It is stated that vaginal fibroids may become sarcomatous, but further proof of this is required before it can be accepted. It is more probable that such growths are sarcomatous from the commencement.

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[1] D'Arcy Power, *Trans. Path. Soc.* vol. xlvii. 1896, p. 169. *Id. St. Bartholomew's Hospital Reports*, vol. xxxi. p. 121. Specimen 3030A, St. Bartholomew's Hospital Museum, occurring in a child two years and four months old.

[2] H. Kelly, *Operative Gynæcology*, vol. i. p. 253.

CHAPTER III

ON PELVIC INFLAMMATION[1]

A SATISFACTORY description of this condition is one of difficulty, though much has been done of late to add to our knowledge of the subject, and many of the ideas and teachings of former years have been proved to be erroneous chiefly by a closer study, not only of the anatomy and pathology of the female pelvic organs, but by the advances of modern abdominal surgery. Pelvic inflammation includes pelvic peritonitis and pelvic cellulitis (or perimetritis and parametritis), the latter words being the names given to such inflammations by Virchow and Matthews Duncan.[2] Such terms will probably last, but from their similarity 'perimetritis' and 'parametritis' are somewhat puzzling words to students, and they imply a difference in the anatomical relations of the peritoneum and of the connective tissue to the uterus, which does not exist, for they are equally in close contact with the uterus, and it is not correct to speak of 'perimetritis' as an inflammation round the uterus, or of 'parametritis' as an inflammation near it. Of course the words have gradually been used with different significance, and to embrace wider conditions; but, as Cullingworth[3] points out, the words 'pelvic peritonitis' and 'pelvic cellulitis' are more correct terms.

Nothing, perhaps, has added more to our knowledge of a very difficult subject than the excellent work that has been done in the study of the anatomy of the female pelvis by frozen sections;[4] while the pathology of Diseases of the Tubes is better understood, and, further, clinical examination has been verified by careful post-mortem examination in fatal cases. At the present time many of the views held by former writers have been found, from closer and more exact scientific observation, to be quite erroneous, or at least unsatisfactory. This is seen in the fact that in former times a large number of swellings in the pelvis associated with pelvic inflammation were termed parametritis, whereas parametritis is practically known to be a somewhat uncommon disease, while perimetritis (or pelvic peritonitis) is common enough, and it was not till proper post-mortem and operative evidence was brought to bear on the subject that such swellings formerly thought to be in the cellular tissue were found to be the result of inflammation of the pelvic viscera and the surrounding peritoneum. Much was done by Bernutz and Goupil[1] on this subject. They pointed out that inflammation of the pelvic peritoneum[2] was a disease frequently met with, that the tumour found after death in cases of pelvic peritonitis was formed by the matting together of various pelvic viscera, and that inflammation of the pelvic serous membrane is generally secondary to inflammations of the Ovaries and Fallopian Tubes.

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[1] For a complete list of literature on this subject see Cullingworth's article 'Pelvic Inflammation,' Allbutt and Playfair, *System of Gynæcology*, 1896, p. 522.

[2] Matthews Duncan, *Perimetritis and Parametritis*, Edin. 1869.

[3] Cullingworth, Allbutt and Playfair, *op. cit.* p. 486.

[4] Berry Hart, *Atlas of Female Pelvic Anatomy*, Edin. 1884; *id. Sectional Anatomy of the Female Pelvis*, Edin. 1885; *id. Selected Papers in Gynæcology and Obstetrics*,' Edin. and Lond. 1893.

Polk's[3] records of operative and post-mortem work at the Bellevue Hospital are worthy of consideration. He points out the rarity of cellulitis and the frequency of peritonitis, and that swellings felt by clinical examination before operation were inflamed tubes and thickened peritoneum, and not swellings of the cellular tissue at all; and further that in such cases the tubes were frequently inflamed, the focus of the peritonitis occurred about the ends of such tubes, and from this focus the inflammation spread to adjacent areas of the peritoneum. Thus, according to Polk the post-mortem evidences went to show that peri-uterine inflammation was a product of salpingitis and not secondary to cellulitis.

Therefore, from a more modern standpoint[4] and on definite evidence, we must conclude that masses in the pelvis formerly described as parametritic are far more frequently due to pelvic peritonitis, and that they frequently consist of diseased Fallopian Tubes, surrounded by thickened peritoneum, involved in a mass of adherent viscera, including almost always the ovary and broad ligament, and, not infrequently, pieces of omentum and one or more coils of intestine.

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[1] Bernutz and Goupil, *Archiv. Gén. de Médecine*, 1862.

[2] Cullingworth, *Clin. Journ.* Aug. 30, 1893, p. 280.

[3] Polk, *Trans. Assoc. Amer. Phys. Philad.* 1886, vol. i.; Cullingworth, 'The Differential Diagnosis of Pelvic Inflammation in the Female,' *Brit. Med. Journ.* vol. ii. 1890, p. 1468.

[4] Cullingworth, *Brit. Med. Journ. loc. cit.*

Another group of cases are associated with pathological pelvic products, such as inflamed ovarian cysts or uterine fibroids; thus pelvic peritonitis, as will be seen later, is in many cases symptomatic and secondary to some disease which already exists.

Unfortunately, with our increase of pathological knowledge came an increase of operative fervour in connection with it, and the word 'salpinx' seemed to become almost a pass-word to open the abdomen, for cases for which it was utterly unjustifiable, and ovaries and tubes were, and are, removed which by less serious procedure might have got quite well. Reference to this will be made later, but the subject is admirably summed up by Champneys[1] in his article on the 'Removal of the Uterine Appendages.' Perhaps the question is apart from a mere work on pathology, but the writer makes no apology for its quotation and for upholding the views therein contained.

The anatomical distribution of the peritoneum of the female pelvis[2] is as follows:—In front it is reflected from the anterior abdominal wall on to the fundus of the bladder, just above the symphysis pubis, it then passes down the posterior surface of the bladder as far as the level of the os internum, whence it is reflected upwards on to the anterior surface of the uterus forming the vesico-uterine pouch. After covering the fundus uteri the peritoneum passes downwards and backwards over the posterior surface of the body of the uterus and cervix, and thence on to the upper fourth of the posterior vaginal wall. It is then reflected on to the front of the rectum and posterior pelvic wall, forming the lowest portion of Douglas's pouch, which is bounded laterally by the so-called utero-sacral ligaments[3] which stretch from the lateral margins of the supra-vaginal cervix upwards and backwards to the sacrum about the level of the second sacral vertebra. The supra-vaginal cervix forms the anterior boundary of Douglas's pouch; posteriorly lies the rectum.

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[1] Champneys, *St. Bartholomew's Hospital Reports*, vol. xxix. 1893, p. 45.

[2] Cullingworth, 'Differential Diagnosis of Pelvic Inflammation in the Female,' *Brit. Med. Journ.* vol. ii. 1890, p. 1468; Herman, 'Contribution to Anatomy of the Pelvic Floor,' *Trans. Obstet. Soc. Lond.* vol. xxxi.; Berry Hart, *op. cit.* Edin. 1885; Barbour, 'Anatomy of Labour as studied in Frozen Sections, and its bearing on Clinical Work,' 1887 and 1896.

[3] The folds of peritoneum should be called utero-rectal; below them is cellular tissue; this may be infected by inflammatory processes or malignant disease; they can then be felt easily by rectal examination, and we say the 'utero-sacral' ligaments are involved, but in health only a little unstriped muscle and cellular tissue in the form of a 'ligament' can be demonstrated by dissection.

Laterally the pelvic peritoneum is prolonged outwards from the lateral borders of the uterus, in the form of folds known as the Broad Ligaments, to the lateral wall of the pelvis on either side. Along their upper border is enclosed the Fallopian Tube, which runs from the uterus outwards to within a short distance of the side of the pelvis, where it leaves the broad ligament to turn backwards, downwards, and inwards, the fimbriæ embracing the outer surface of the ovary. The ovary lies behind the broad ligament, and below the external iliac vessels on a lower plane than the tube, resting in a depression called the 'Ovarian Fossa.'[1] The special fold of peritoneum belonging to the Fallopian Tube is known as the meso-salpinx. Beneath the ovary the layers of the broad ligament separate, passing to the anterior and posterior pelvic walls respectively. The broad ligaments thus divide the pelvis transversely, and the spaces in front and behind may be spoken of as the anterior and posterior quarters of the pelvis right and left. The anterior quarters are shallow and unimportant, and contain no important organs, while the posterior contain the free ends of the Fallopian Tubes and the ovaries; and since Douglas's pouch lies between, in close relation with the upper part of the vagina, the ovaries and tubes can in this situation be more or less easily reached by clinical examination. The importance of the posterior quarters of the pelvis in relation to pelvic peritonitis cannot be too strongly insisted upon. In health the outline of the pelvic peritoneum is not perceptible to the touch, and on vaginal examination no resistance is offered beyond the tension of the vagina; but when the peritoneum is thickened by inflammation, its shape can be felt dipping down into Douglas's pouch or at the sides, as an induration joining the uterus at the level of the os internum, and then sloping upwards towards the pelvic wall. Sometimes this thickened peritoneum, on vaginal examination, feels as though it were the lower surface of a large, solid swelling, and it is only on careful bimanual examination that we can determine it to be simply thickened peritoneum of the floor of the pelvis.

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[1] Cripps, *Ovariotomy and Abdominal Surgery*, 1898, p. 31.

The Cellular Tissue of the Pelvis lies beneath the peritoneum and forms a matrix in which all the pelvic organs are embedded. It fills the lower part of the pelvic cavity except where the urethra, vagina, and rectum perforate it. Imagine the pelvic organs to be quite bare and placed in the pelvic cavity, and then, as it were, dredged with flour till they were more or less covered in. Imagine some of the flour collecting at the sides, in front of, and behind the uterus, in ridges of more or less depth. Let these be in the regions of the vagina and bladder in front, the broad ligaments laterally, around the cervix, and thence, extending by the utero-sacral ligaments backwards, towards and around the rectum, the whole resting upon the floor of the pelvis; then imagine this covered all over everywhere by a thin sheet of gutta-percha tissue, and one has a rough simile of the cellular tissue distribution (the flour), and peritoneum (the gutta-percha tissue), investing the pelvic organs.

All the pelvic cellular tissue is above the pelvic fascia covering the levator ani muscle. The cellular tissue of the vulva and ischio-rectal fossa is quite distinct, being below this fascia.

At the sides of the pelvis the cellular tissue is continuous with the general subserous connective tissue. Through the inguinal and femoral canals it is continuous with the cellular tissue of the thighs, and by the sciatic foramina with that of the buttock. In the pelvis, as regards the viscera it forms a layer beneath the entire peritoneum, but (as in the simile given above of the flour), it is more abundant in certain areas. It is particularly marked around the supra-vaginal cervix, along the base of the broad ligaments, between the bladder and symphysis, and around the rectum. Around the uterus it is close in texture, and over the greater part of the fundus scanty, and the peritoneum is closely attached to it. Below, and surrounding the supra-vaginal cervix, there is a very important area of cellular tissue continuous in front with that of the bladder. On each side, the cellular tissue extends into the broad ligaments and is very well marked; fat also occurs in this situation. On section this mass of connective tissue in the broad ligament forms a triangular wedge of tissue having the Fallopian Tube at its summit, the wedge increasing in size towards the pelvic floor; in this highly important layer of connective tissue run the vessels, nerves, and lymphatics of the uterus. The round ligament, and ovarian ligament, also traverse it. The parovarium and other developmental relics are described in connection with Ovarian Tumours. Posteriorly, important tracts of cellular tissue are seen in the utero-sacral ligaments which bound Douglas's pouch, and pass backwards to be continuous with the peri-rectal connective tissue. Thus, as Herman[1] points out, the cellular tissue of the pelvis is somewhat star-shaped in its distribution, spreading from the uterus as a centre, and radiating forwards, backwards, and laterally, as above described. Each limb thus reaches the pelvic wall; this explains the spread of cellulitis, which of course depends on the continuity of cellular tissue.[2]

Difference between the Pregnant and Non-Pregnant State.—Pregnancy[3] greatly alters both the amount and relations of the cellular tissue of the pelvis, and this fact cannot too strongly be insisted upon. The amount is vastly increased especially at the base of the broad ligaments, which are lifted up by the growing uterus, so that at the end of the pregnancy the base of the broad ligament is really at the brim of the pelvis. Beneath the base of the broad ligament is a large triangular area, at the side of the uterus, uncovered by peritoneum, which as pregnancy advances becomes filled with new cellular tissue. Continuous with this is a large area of cellular tissue behind Poupart's ligament on either side, as the vesico-uterine pouch now no longer dips into the pelvis (as in the unimpregnated state), nor does any peritoneum dip into the lateral parts of the pelvis. This lifting up of the peritoneum by the growing uterus, and the vast increase of connective tissue which fills the gaps of continuity between it and the pelvic floor, and which surrounds the cervix below, plays a most important part in the course and situation of cellulitis occurring after delivery. Instead of the inflammation remaining merely pelvic as in the impregnated state, after delivery the loose cellular tissue above the brim is affected and a swelling forms (above the brim) behind Poupart's ligament. This alteration in the disposition of the pelvic peritoneum and cellular tissue as the result of pregnancy explains the curious position of some of the inflammatory swellings occurring after delivery, which now appear as abdominal instead of pelvic, and which are sometimes very puzzling in diagnosis.

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[1] Herman, *Diseases of Women*, p. 256.

[2] Cullingworth, *Brit. Med. Journ.* Dec. 27, 1890.

[3] Herman, *Diseases of Women*, p. 257; Barbour, *op. cit.*

Continuity of the Pelvic Cellular Tissue.[1]—As above stated, the pelvic cellular tissue is continuous with the subserous connective tissue. Vessels, nerves, and lymphatics are embedded in it, and tracts of cellular tissue accompany them in their exit from the pelvis, *e.g.* into the buttock, with the great sciatic nerve, with the obturator vessels, down the crural canal, and along the round ligament. Again there are special tracts of cellular tissue covering the iliacus, tranversalis, and psoas muscles,[2] which are in continuity with the peri-nephric connective tissues above. Other tracts surround viscera, notably the rectum, which in cellulitis is sometimes encircled with a dense fixed collar of inflamed tissue. The fixity of the cervix in pelvic cellulitis, the dense infiltration of the base of the broad ligament, and the involvement of the connective tissue around the bladder can thus be easily explained, if the anatomical distribution of the cellular tissue be kept in mind.

Pelvic Peritonitis (Perimetritis).—In the vast majority of cases pelvic peritonitis is set up by septic absorption through the uterus and tubes to the peritoneal cavity. Frequently the lining membrane both of the uterus and tubes is involved; this may or may not be evident; but the infection once reaching the peritoneum, a localised peritonitis is established in one or other posterior quarter of the pelvis, around the mouth of the tube. We get clinical evidence of the local peritonitis in the pain which accompanies it, though the mere endometritis or salpingitis have not drawn attention to the case. Still the importance of Diseases of the Tubes[3] in relation to Pelvic Peritonitis cannot be over estimated.

Causes of Pelvic Peritonitis.

a. Septic absorption after labour, abortion, or the use of dirty instruments or tents.
b. Gonorrhœa.
c. Simple catarrh.[4]

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[1] Cullingworth, *op. cit. Brit. Med. Journ.* vol. ii. 1890, p. 1468.

[2] Some authors even give special names to varieties of Parametritis according to the tracts affected, *e.g.* 'Psoas' Parametritis, 'Iliac' Parametritis, and so on.

[3] Cullingworth, *op. cit.*

[4] Many authors deny the possibility of 'simple catarrh.'

d. Inflammation of pelvic organs and pathological products, such as ovarian cysts, fibroids, diseased tubes or extra-uterine sacs, hæmatocele, and, rarely, malignant disease.

With regard to **labour and abortion** or the use of septic instruments, peritonitis is secondary to disease of the uterus, the infection reaching the peritoneum through the tubes. The same remarks apply to gonorrhœa. **Simple catarrh** may be a milder form of infection often difficult to trace, and not easy to determine under the popular heading of 'chills and catching cold'; in fact, cases of pelvic peritonitis due to catching cold must be looked upon with suspicion, though it is possible to imagine a catarrh of a simple nature spreading to the tubes and peritoneum from the uterus. The infection of **gonorrhœa**[1] will be discussed under a separate heading, but its importance in the etiology of pelvic peritonitis is certainly very great.

With regard to the **pathological pelvic products**, we must distinguish between cause and effect. Papilloma of ovary or tube, an ovarian tumour with a twisted pedicle or a dermoid cyst, a suppurating extra-uterine sac, hæmatocele, a degenerate or suppurating fibroid, a diseased appendix vermiformis, malignant disease of the uterus or bowel, may be associated with pelvic peritonitis. It is rather with the main disease than with the mere peritonitis that we are concerned, both in diagnosis and treatment in such cases, though in some cases of pelvic peritonitis it is not till the acute peritonitic attack has passed off that we can discover some pathological product in the pelvis as its cause. In the vast majority of cases, pelvic peritonitis is **septic**; it is perhaps the commonest disease of women, and much commoner than cellulitis. Its results are limited in most cases. and, in a way, it is a conservative process,[2] the adhesions limiting its spread. The acute infective cases, as seen in Puerperal Fever, are too rapidly fatal for any such conservative action to be set up,[3] and are frequently accompanied by diffuse cellulitis as well.

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[1] Cullingworth, 'The Etiological Importance of Gonorrhœa in relation to the more common Diseases of Women,' *Brit. Med. Journ.* vol. ii. 1889.

[2] Treves, *Peritonitis*, London, 1894.

[3] Targett, 'Acute Suppuration and Sloughing of the Ovaries after Parturition,' *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 216.

The greatest interest centres upon the relation of pelvic Peritonitis to Diseases of the Tubes, as in a great proportion of cases such disease exists, and Peritonitis is a constant sequel of Salpingitis. In fact, many authorities cease to give pelvic peritonitis a primary place, but regard it as an indication of some other disease, and not one by itself; and certainly the results of post-mortem investigation and operative treatment of late have added much to our knowledge of this subject, especially in chronic recurring cases.

With regard to the condition of the Tubes themselves in Pelvic Peritonitis more will be said (see 'Diseases of the Tubes'). Here it will suffice to say that the tube may or may not be closed and its secretions retained, and that the inflammation may either spread along the tube or through its walls, or the walls give way by ulceration as in pyosalpinx, though this is very rare. The ovary may be inflamed as well, generally secondarily, and may suppurate, especially if such an ovary be cystic. An abscess of the ovary may occur, or the contents of a suppurating tube may enter a cyst in the ovary (tubo-ovarian abscess). The pus in such cases may escape into the general peritoneal cavity,[1] giving rise to peritonitis; much more commonly it is cut off by adhesions, and a limited intra-peritoneal abscess is formed.

Pathological Anatomy.[2]—The first evidence of peritonitis is hyperæmia; the peritoneum loses its shiny smooth appearance and becomes dull and roughened, the epithelium swells, lymph is poured out and adhesion results. In fact we might compare pelvic peritonitis with pleurisy in its stages, and give, as a classification, three *varieties*:

1. Adhesive Perimetritis.
2. Serous Perimetritis.
3. Purulent Perimetritis.

Adhesions are of vast importance in pelvic peritonitis, as they tend to limit the spread of the attack. At the commencement we find the adhesions only around the mouth of the tube; later they may even close the end of the tube (perimetritic closure)[3]; next, the ovary, tube, and intestines, in

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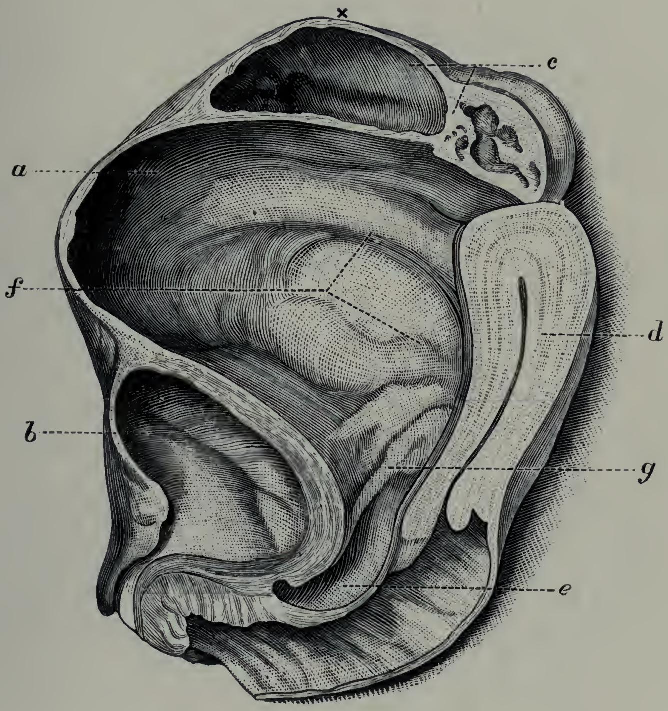
[1] Lewers, 'Double Pyosalpinx with Rupture of the Tubes,' *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 298.

[2] Cullingworth, 'Pelvic Inflammation,' Allbutt and Playfair, *op. cit.* p. 503.

[3] Doran, Allbutt and Playfair, *op. cit.* p. 792.

Fig. 9.

ANTERIOR PERIMETRITIS.



Specimen of anterior perimetritis (pelvic peritonitis) in sagittal section, situated in the utero-vesical pouch and simulating an inflamed ovarian cyst.

a. Abscess cavity.
b. Bladder.
c. Right ovary (cystic).
d. Uterus.
e. Extension of abscess into anterior parametric region.
f. Structures of right broad ligament laid bare.
g. Right ureter, exposed.
× Fimbriated extremity of right tube adherent at this spot.

(Griffith, 'Trans. Obstet. Soc. Lond.' vol. xxix. 1887, p. 149.)

the neighbourhood are matted and glued together into an indistinguishable mass, and finally the whole pelvis is roofed in by adhesions which limit the spread of the infection to the general peritoneal cavity. This explains the rarity of general peritonitis.

The lymph in perimetritis may be of considerable thickness and amid the coils of intestine curious lymph coagula are sometimes formed, giving rise to a definite swelling; at other times there may be collections of serum; in fact, such serum surrounded by peritoneal adhesions may assume the clinical characters of an ovarian cyst. The name 'Serous Perimetritis'[1] is given to such a condition. The serum and lymph may be eventually absorbed, but the adhesions remain, and may cause serious trouble later on.

Serous peritonitis is important when the serous fluid effused is in large amount. In ordinary adhesive peritonitis it is common to get spaces between adhesions containing a little fluid; large effusions of serum are not common. Why one case of peritonitis should be associated with much effusion and another with little is unknown; abscess of the ovary is sometimes a cause. Large effusions shut up in adhesions are important because they cause great tension, pressure,[2] and displacement of the parts either in the pelvis or above it. The swelling itself[3] may cause great difficulty in diagnosis, but such cases are preceded by inflammation; they are fixed and often surrounded by adherent bowel. It is generally a severe variety of pelvic inflammation, and leads to great prostration with fever, sometimes ending fatally. The false capsule surrounding such an effusion is often very thick, and may even resemble a cyst wall till its relations are demonstrated by operation or dissection. Effusions of this nature have been mistaken for ovarian cysts (fig. 9), intra-peritoneal abscess, and hæmatocele.

Targett described a case[4] in which the effusion in serous perimetritis formed a tumour reaching half-way to the umbilicus. The greater part of the mass at the operation consisted of a multilocular cystoma, formed between adhesions in connection with the left uterine appendages, the spaces being filled with clear serous fluid ; beneath it was the thickened and distended left tube containing pus. The boundaries were, in front, the stretched meso-salpinx and broad ligament of the left side ; below, and on the outer side, the left ovary and distended tube ; above it was the uterine end of the left tube and adherent omentum, while anteriorly it was bounded by adherent sigmoid flexure. A large space was thus formed, which was subdivided by membranous adhesions into loculi, and the whole space was filled with 8–10 oz. of serous fluid. Similar collections of fluid are by no means uncommon in cases of chronic salpingitis, though not usually so large. It is important in such cases to recognise that such fluid collections are not between the layers of the broad ligament, but in the peritoneum, generally behind one or other broad ligament, and shut off by adhesions which form between the various organs in consequence of local perimetritis. Very frequently the firmest adhesions are met with near the mouth of the tube, binding it to neighbouring parts.

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[1] Cullingworth does not think that 'serous perimetritis' is a special variety of pelvic inflammation: Allbutt and Playfair, *op. cit.*, note at foot of p. 503.

[2] The pressure may distend Douglas's pouch, interfere with the bowel or bladder, or cause elongation of the cervix, or even sloughing of cervix, vagina, and rectum.

[3] Targett proposed the name of 'perimetric cystoma' for such an encysted collection of fluid, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 343.

[4] *Op. cit.* p. 343.

Purulent Perimetritis. Perimetric Abscess.—The case may go on further, and, instead of adhesions and serum, pus forms in the pelvis. In many cases the tube or ovary is the starting point, and the tube may itself contain pus ; generally, when the tubes are at fault, they soon contract adhesions to the ovary and back of the broad ligament, and are found deep in Douglas's pouch, unless lifted out of the brim by pregnancy.[1] They are bent upon themselves, and have the usual characteristic retort-shape; such tubes may even open into one another. The ovary is rarely healthy, and in puerperal cases abscess is not uncommon. Pelvic abscess may depend on a pathological pelvic product, *e.g.* a suppurating ovarian cyst, especially a dermoid, which is prone to inflame, or a cyst in which the pedicle has become twisted, and which may then undergo inflammatory and suppurative changes. In other cases pus forms around a sloughing or gangrenous fibroid, or extension of inflammation from the bowel occurs in appendicitis or cancer. Sometimes with cancer of the uterus, especially of the body, large collections of pus may form. At other times the abscess is due to breaking down of lumbar glands, or tubercular conditions of the tube are present.

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[1] Cullingworth, Allbutt and Playfair, *op. cit.* p. 505.

The formation of pus in connection with a suppurating pelvic hæmatocele is important, and will be discussed under Extra-uterine Pregnancy.

In some cases of pelvic abscess, when inflammation has subsided in the pelvis, suppuration goes on in another part of the pelvic cavity. Such cases are termed 'remote.'

It is important to remember that in puerperal cases the abscess may be entirely above the brim, though it may subsequently find its way into Douglas's pouch.

Course of Perimetritis.

a. May get quite well.
b. May end in abscess.
c. May become chronic or recurrent.

The majority of cases of slight pelvic peritonitis recover, and the disease itself is one of low mortality, as witnessed by the frequency of its occurrence in women, and the infrequency of cases seen in the post-mortem room. Cases of fatal peritonitis due to the bursting or ulceration of pus-containing tubes into the general peritoneum *are very rare*, but a few are on record.[1] In nearly all cases, whether the suppurating focus be in the tube or not, the general peritoneal cavity is shut off by adhesions. The pus may ultimately be absorbed, or may escape into adjacent viscera,[2] generally the rectum or sigmoid flexure, rarely into the bladder or vagina. The common way of escape of the contents of an intraperitoneal abscess is undoubtedly by the bowel. The case may then get well, but troubleseme sinuses may remain and discharge for years. In such cases a definite pathological product probably exists, such as a suppurating tube or ovarian cyst. The continued discharge of pus may lead to lardaceous disease and great wasting.

Chronic and Recurrent Cases.—These are rare, but sometimes very troublesome, and it is here that such good results have accrued from operative treatment, though, as before stated, the great majority of such cases get well. In others, adhesions may form and the uterus may be more or less fixed, but such adhesions may not cause any symptoms. The mere fixation of a uterus, or even its flexion, is not important; a uterus a little to the right or left is not a disease in itself, though it is often wrongly treated as such. The fixation of a uterus in Douglas's pouch with tender inflamed appendages after an attack of perimetritis may be important, and the bowel or omentum may be similarly bound down, giving rise to chronic pelvic pain, which, in spite of all local treatment and long periods of rest in bed, will still persist. Hydrosalpinx sometimes occurs with such chronic cases.[1] Hæmatosalpinx is very rare.

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[1] Lewers, *Trans. Obstet. Soc. Lond.* vol. xxvii. p. 298 and vol. xxix. 1887, p. 199; Cullingworth, 'On the Value of Abdominal Section in certain cases of Pelvic Peritonitis,' *Trans. Obstet. Soc. Lond.* vol. xxxiv. 1892, p. 254, and discussion, Williams, Champneys, *ibid.* pp. 430–442; Grigg, *Journ. Brit. Gyn. Soc.* vol. ii. p. 264.

[2] Cullingworth, Allbutt and Playfair, *op. cit.* p. 505, 506.

Another set of cases in which relapse and recurrence take place are those in which the pelvic peritonitis depends upon a cause which still remains, *e.g.* the pathological pelvic products mentioned above, especially the chronic cases of suppurating tubes. Pain in peritonitis is due to movement of the inflamed parts, especially the peritoneum; the bladder is frequently irritable, and the bowel also. Intestinal obstruction is very rare, but constipation should always be guarded against. Fever accompanies perimetritis in acute cases, and in the relapsing and recurrent ones, but it is well known that we may get intraperitoneal suppuration with a normal temperature.

Sterility in perimetritis must necessarily result if the tubes are closed and this probably occurs in a large majority of cases, yet the prognosis of sterility in perimetritis must be a guarded one. Many cases even after a severe attack become pregnant. Lardaceous disease is seen in cases of prolonged suppuration, sometimes with emaciation equal to that of malignant disease; besides this, the woman may become a hopeless invalid, and neurasthenia is frequent.

Cases may completely recover from these symptoms and yet some lump may remain in the pelvis; in fact, some thickening in the pelvis of a woman who has borne children can frequently be found, yet it causes her no trouble. Such lumps do not need operation, they do not kill, in fact the prognosis of perimetritis is very good indeed, its mortality is low, removal[2] of the appendages is rarely needed, and the limits of the justifiability of operation rare, as an uncomplicated disease. Of course, if diseased tubes, ovarian cysts, or other products persist, it is another matter, as such conditions certainly justify operative procedure, and it is particularly with regard to these chronic suppurating tubes[1] that so much light has been thrown lately by operative measures and with such good results; but such are not to be lightly undertaken, for the mortality of bad operations is high indeed.

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[1] Doran, 'Changes in the Ostium,' Allbutt and Playfair, *op. cit.* p. 791.

[2] Champneys, 'On Removal of the Uterine Appendages,' *St. Bartholomew's Hosp. Reports*, 1893, p. 45.

Pelvic Cellulitis—Parametritis.[2]—This, as a primary disease, is always septic, and spreads directly outwards from the cervix or body of the uterus to the cellular tissue. There is no involvement of the tubes in cellulitis.

In nearly all cases there is some laceration of the cervix or vagina, frequently by instruments in labour and abortion, or the use of tents. The mere injury is not, of course, the cause of the cellulitis, which is due to sepsis, but bruising and tearing of the tissues renders them more liable to infection, while the tears themselves may expose the cellular tissue of the broad ligaments to direct absorption. Lymphatics in all cases convey the septic material; in fact, in many cases there is a pelvic lymphangitis; glandular enlargement in the pelvis cannot be made out, though it probably occurs. Pelvic cellulitis as the result of infection is rarer than peritonitis.

Referring to the anatomical distribution of the cellular tissue, and especially to the vast increase in connection with pregnancy, the course and results of cellulitis will be better understood.

Pathological Anatomy.[3]—In a case of cellulitis starting from the **body** of the uterus, the inflammation is transmitted directly through the uterine tissues to the cellular tissue and connective tissue between the layers of the upper part of the broad ligament, limited at first to that portion adjacent to the uterus. It then travels to the lateral wall of the pelvis, or forwards beneath the anterior layer of the broad ligament to the sub-peritoneal connective tissue of the anterior abdominal wall, or, having reached the lateral wall of the pelvis, the inflammation may pass either upwards along the psoas muscle to the region of the kidney, or, more commonly, downwards and forwards to the anterior abdominal wall, creeping along above Poupart's ligament to the mass of fat and cellular tissue lying in front of the bladder and above and behind the symphysis. This same spot is reached by the inflammation if it travels forwards instead of laterally; thence it may continue to spread upwards beneath the anterior abdominal wall, even as high as, or higher than, the level of the umbilicus. In the case of cellulitis originally in the cervix, the cellular tissue immediately surrounding the cervix is the first to be affected; the inflammation then spreads along the base of the broad ligament to the wall of the pelvis; it may also affect the vesico-uterine cellular tissue in front, or the utero-sacral ligaments behind, in which latter case the rectum will be surrounded by exudation on at least three of its sides, and in some instances will be completely encircled.[1]

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[1] Cullingworth, 'On Pelvic Peritonitis in the Female, and the pathological importance of the Fallopian Tubes in connection therewith,' *Clin. Journ.* No. 18, vol. ii. p. 279.

[2] Cullingworth, 'Differential Diagnosis of Pelvic Inflammation in the Female,' *Brit. Med. Journ.* vol. ii. 1890, p. 1468; Herman's 'Lectures on Parametritis,' *Clin. Journ.* vol. vi. Nos. 9–12, 1895; and Herman, *Diseases of Women*, p. 254, article 'Parametritis.'

[3] Herman, *op. cit.*, p. 260

Parametritis, in a woman after labour, usually spreads into the loose cellular tissue above the brim already described, and forms a swelling high up in the pelvis behind Poupart's ligament. Parametritis in this situation is rare in a woman not recently delivered.

If the poison be very virulent, as is seen in cases of Puerperal Fever, the condition is one of diffuse œdema like phlegmonous erysipelas, sometimes with suppurating foci in the cellular tissue, or veins and lymphatics may be involved. Ovarian suppuration and peritonitis also occur; such forms are very fatal.[2]

Frequently parametritis takes a less severe form, and may occur with or without the formation of pus; in mild cases exudation and œdema of the cellular tissues are the chief factors. In cases where infection follows on a laceration of the cervix the swelling is one-sided, smooth and uniform, depressing the vaginal roof; often, it may be very hard, or a distinct collar of induration surrounds the cervix, causing great fixity. If the broad ligament be involved, the cellulitis expands and fixes it, depressing the vaginal fornix on that side, forming a very definite swelling, spreading out to the sides of the pelvic wall. If there is a swelling behind Poupart's ligament, it has definite limits.

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[1] Cullingworth, *op. cit. Brit. Med. Journ.* vol. ii. 1890, p. 1468; see also Herman, *Diseases of Women*, p. 262, and figs. 88, 89, 90, pp. 263, 264.

[2] Lewers, 'Post-mortem Appearances of a Phlegmon of the Broad Ligament,' *Trans. Obstet. Soc. Lond.* vol. xxx. 1888, p. 7.

Cellulitis is characterised by the extreme hardness and induration of the tissues, in spite of which **resolution** may take place, and practically no trace of the attack remain, or merely some scar tissue is left, more or less fixing the uterus and drawing it to one side.

Section of the tissues in cellulitis finds them spongy and œdematous, while in the acute puerperal cases there may be multiple pus foci, and even hæmorrhage into cellular tissue. Acute cases of this nature are associated with perimetritis as well.

While by far the greater number of cases of parametritis are puerperal in origin, some are caused by injuries with septic instruments, tents, dilators, or stem pessaries ; and before antiseptics were used, and their use understood in gynæcology, disastrous effects followed many such operative procedures.

New growths involving the uterus, such as cancer of the cervix, especially with foul ulcerating cavities, are also associated with cellulitis, which may cause fixity, and the infiltration is taken to be malignant.

Cellulitis may end in **abscess**, and it does so in a considerable number of acute cases. Most cases of cellulitic abscess point above Poupart's ligament, near the internal ring where the round ligament leaves the abdomen, a soft spot appearing in the centre of the induration in that region. (Intra-peritoneal abscess never points there.) Rarer situations of bursting are into the bladder and vagina. Abscesses which point, or are allowed to burst, above Poupart's ligament, are sometimes very slow in pointing and troublesome in healing, the pus continuing to discharge for long periods, probably from the deeper parts of the cavity, which may consist of several loculi communicating, though there is generally only a single one.

Rarer Situations of Pointing.[1]—Referring again to the anatomical distribution of the pelvic cellular tissue, parametric abscesses may point in unusual situations. Thus perinephritic suppuration has occurred, the pus has tracked into the iliac fossa, or down the thigh, or even discharged through the umbilicus or mid-line of abdomen. Following vessels, the abscess may point in the gluteal region, down the thigh, or even along the obturator internus in the region of the great trochanter.

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[1] Herman, *Diseases of Women*, p. 274.

It is rare for a cellulitic abscess to burst into the rectum, most of such cases being intra-peritoneal (perimetric).

The vast majority of parametric abscesses, however, point above Poupart's ligament.

Varieties of Parametritis.—Those in connection with *puerperal infection* form the greater number (Herman); they are all septic. The most acute cases, which are fortunately rare, resemble an acute phlegmonous erysipelas, and are rapidly fatal. The ordinary puerperal case, where the broad ligament is affected and a definite lump felt close to the cervix, spreading out to the sides of the pelvis, may end in resolution and complete recovery. As a rule, about half such cases run on to suppuration, with the ordinary swelling above Poupart's ligament limited by the adhesion of the transversalis fascia to the peritoneum, or the pus may track in any of the situations above described. If resolution occur, adhesions may still remain, dragging the uterus over; it is not common for such adhesions to involve important structures, but sometimes the ureter, pelvic nerves, or the rectum, are involved in the scar tissue.

The chief distinguishing factor of puerperal cases is the tendency for all the inflammation to be situated above the brim.

Non-puerperal Parametritis[1] occurs, but is less common than puerperal; it is septic, and often due to lacerations made by unclean instruments or tents. The inflammation is limited to the pelvis, does not travel about the brim, and takes the form of infiltration and phlegmon. The infiltration of the tissue is often very hard, dense, and fixed, especially about the utero-sacral ligaments, and around the rectum. It may remain for a long time. Abscess is uncommon, it may point at the sides of the sacrum, or into the vagina or rectum, or the pus may remain encapsuled for a long period.

Chronic Parametritis is probably not the sequel of any acute form; a chronic disease should have a chronic cause, and, in most cases, such exists in the pelvis as a constant source of irritation. Some cases seem to be connected with breaking down glands, or malignant disease of bowel, bladder, or uterus, or even necrosis of the pelvic bones. It appears to be a more or less independent affection, and takes the form of infiltration and œdema, for pus rarely forms. Matthews

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[1] Herman, *Diseases of Women*, p. 267.

Duncan described a form termed 'Parametritis Atrophicans,' which he thought occurred as a sequel to the acute form.

Freund[1] thinks that the disease is chronic from the first, and that the disease depends upon a chronic pelvic lesion; the whole or part of the pelvic cellular tissue may be affected, and the hardness and fixity are remarkable; little change takes place, the condition remaining the same even for years. The disease has important bearings, in that important structures are embedded in the cellular scar tissue, such as veins or ureters, or the lumen of viscera may be affected, leading indeed to a form of stricture.

Matthews Duncan[2] also describes 'Hæmorrhagic Parametritis,' where, in the course of an acute spreading infective cellulitis vessels may be opened by sloughing. This is the same disease as described above under acute puerperal parametritis. Remote parametritis has already been referred to.

Anterior Parametritis.—This form is rare,[3] the cellulitis being limited to the loose vesico-uterine cellular tissue, or that between the symphysis and the bladder; it has occurred after the operation of symphysiotomy. The swelling is anterior, and the pus generally tracks into the bladder, vagina, or inguinal region. It is likely to be mistaken for an ovarian cyst.

Parametritis and Albuminuria.—This was first described by Matthews Duncan[4] and occurred in a considerable number of cases. In some it appeared to be due to cystitis, or pus bursting into the bladder or urinary tract, or where ureters were surrounded with cellulitic deposit; in other cases no such cause existed. Duncan thought the kidney shared the inflammation of the generative tract in some cases, or the inflammation might extend to the kidney and be in a sense 'remote.'

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[1] *Gyn. Klinik*, Strasburg, 1885, and Herman, *Diseases of Women*, p. 280.

[2] *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 191; Herman, *op. cit.* p. 272.

[3] Griffith, nine out of fifty-five cases of parametritis observed. 'Anterior Perimetritis and Anterior Parametritis,' *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 147, and vol. xxx. p. 5. *Id. St. Bartholomew's Hosp. Reports*, 1880, p. 285.

[4] Matthews Duncan, *Med. Chir. Trans.* vol. lxvii. 1884.

CHAPTER IV

GONORRHŒA IN RELATION TO PELVIC INFLAMMATION.

Although gonorrhœa in women [1] is in England perhaps regarded as a somewhat unimportant disease, there is no doubt that it really destroys the health of a larger number of women than does even the virus of syphilis. This may be partly due to the earlier indications of the disease passing unnoticed, as there may be little or no pain till the peritoneum is involved, and the urethral symptoms and discharge may be quite slight; yet the importance of the fact that gonorrhœa produces the most serious and profound changes on the pelvic organs cannot be too strongly insisted on. Cullingworth, Tait, and Sinclair have done much to bring this subject into prominence, as have also Noeggerath and Sänger, whose papers may be referred to. Sinclair's work gives all the most important Continental references on the subject, and he points out that little attention is paid to the subject by most writers in English text-books, while the results of gonorrhœa on the community are extremely serious.

Its frequency is difficult to state, as it varies, both in localities and in great cities, within wide limits, so that it is impossible to formulate any statement [2] that can have any general application, and the same applies to the various classes of society. It is certainly a common disease amongst hospital patients. In cases of pelvic peritonitis, there can be little doubt that septicæmia following parturition or abortion is the chief cause, but that next in importance comes gonorrhœa.[1]

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[1] Cullingworth, 'The Etiological Importance of Gonorrhœa,' *Brit. Med. Journ.* vol. ii. 1889, p. 122.

W. Japp Sinclair, *Gonorrhœal Infection in Women*, 1888.

Lawson Tait, *Diseases of Women*, 1877.

Noeggerath, *Die latente Gonorrhöe im Weiblichen Geschlecht*, Bonn, 1872. *Id.* 'Ueber latente und chronische Gonorrhöe beim Weiblichen Geschlecht,' *Deutsche Med. Wochenschrift*, Dec. 8, 1887. *Id.* 'Latent Gonorrhœa and its Influence on Fertility,' *Trans. Amer. Gyn. Soc.* vol. i. 1876 (quoted by Cullingworth).

Sänger (M.), *Amer. Journ. Obstet.* March 1887.

[2] W. J. Sinclair, *Gonorrhœal Infection in Women*, p. 22.

Pathology of Gonorrhœa.—Little attention was paid to the subject till Noeggerath's work appeared in 1872, in which he urged the latency and even the incurability of gonorrhœa; this important paper is referred to frequently by Sinclair,[2] and justly so. Noeggerath was unaware of the existence of a gonococcus, but he drew attention to the great importance of neglected gonorrhœa in men in relation to gonorrhœal infection in women, for some of the very worst cases of acute and recurring perimetritis occurred in the wives of husbands who had been supposed to be cured of gonorrhœa shortly before marriage. Noeggerath subsequently modified his views of the incurability of gonorrhœa.

Another important point was brought forward by Angus Macdonald,[3] in which gonorrhœal infection had given rise to more or less severe puerperal illness. Sänger thinks such puerperal illnesses are later in their onset than in cases originating otherwise.[4]

In 1879 Neisser[5] discovered the gonococcus, staining the pus with a watery solution of methyl violet, and concluded that it was characteristic of the disease, and that it was a distinct organism both functionally and morphologically, also that it occurred in masses or colonies, never in chains in the pus cells. The gonococcus appears to have been easily found in acute cases, but in old-standing ones even of only a few months' duration, most certainly gonorrhœal in their nature, which have been under treatment, it was almost invariably impossible to discover it.

Bumm, of Würzburg,[6] published a very important work on the pathology of gonorrhœal infection, in which he proved the existence of micro-organisms (diplococci) which cannot be distinguished by the individual form alone from Neisser's gonococcus, and it was not till the effect of Gram's[1] iodo-iodide solution and washing with alcohol was discovered, which is said to fix the colour (methyaniline blue in aniline water) in all micro-organisms except the gonococcus, that any definite means of differentiation was known. The question now arises as to the diagnostic value of the gonococcus in a discharge. Sinclair[2] says that 'all experience goes to show that the presence of the gonococcus settles the question ; it is the pathognomonic sign of the disease' ; but that the whole subject is in too inchoate a state to make it safe to allege that these conclusions are sufficient to satisfy the requirements of medico-legal investigation.[3] To sum up, then, the collection of the secretion should be taken from the cervix or urethra, or possibly from the orifices of Bartholin's glands ; and as vaginal gonorrhœa is rare, pus from this source should not be used. The particular arrangement of the diplococcus in the protoplasm of the pus cells or other elements of the fluid are characteristic,[4] while the presence of some clinical sign or symptom may suggest the existence of comparatively recent infection.

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[1] Cullingworth, *op. cit. Brit. Med. Journ.* vol. ii. 1889, p. 122.

[2] W. J. Sinclair, *op. cit.* p. 30.

[3] A. Macdonald, *Edin. Med. Journ.* June 1873, 'Latent Gonorrhœa in Relation to the Puerperal State.'

[4] W. J. Sinclair, *op. cit.* p. 43.

[5] Neisser, *Centralblatt für die Med. Wissenschaften*, 1879, No. 28. *Id. Deutsche Med. Wochenschrift*, 1882, p. 279; Sinclair, *op. cit.* p. 50; see also chapter on 'Micro-organisms in Relation to Diseases of Women' in this work.

[6] E. Bumm, *Der Mikrorganismus der Gonorrhöischen Schleimhaut-Erkränkungen*, '*Gonococcus Neisser*,' 2nd ed. Wiesbaden, 1885; J. F. Bergmann, 1887; quoted by Sinclair, p. 58, *op. cit.*

Further light has been thrown on the question by inoculation and cultivation,[5] and by the results of vaginal discharges of puerperal women on the eyes of newborn children, and though cases of ophthalmia may occur, in which the gonococcus cannot be found, they differ in that the discharge from such cases is not infectious, nor is it followed by the same results.

With regard to cases of vulvo-vaginitis in children[6] much controversy has taken place as to whether the diplococci observed can by any means be differentiated from the gonococcus, and many observers think the disease identical with the gonorrhœa of adults. Robinson,[7] in examining fifty-four cases of discharges in children seen under five years of age, found organisms in the discharges like the gonococcus in 76 per cent., but it seems difficult to believe that the percentage should be so high. The vulvo-vaginitis of children is a difficult subject of investigation, but probably gonorrhœal and non-gonorrhœal cases occur, and, as McCann[1] points out, other diplococci simulate the gonococcus, and he refers to Bumm's paper,[2] but such diplococci will liquefy gelatine, while the gonococcus will not grow on gelatine or liquefy blood serum. The cultivation test of the gonococcus is of great value in diagnosis, and should be applied if possible.

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[1] Gram's method is said to have been discovered by Roux, of Paris : *Le Concours Médical*, Nov. 13, 1886 ; quoted by Sinclair, *op. cit.* p. 62.

[2] W. J. Sinclair, *op. cit.* p. 67.

[3] *Ibid.* p. 66 ; see also McCann, 'Etiology of Gonorrhœa,' *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 257.

[4] Other organisms react to Gram's stain besides the gonococcus. McCann, 'Etiology of Gonorrhœa,' *op. cit.* p. 255.

[5] Experiments of Bumm and Zweifel, quoted by Sinclair, p. 67.

[6] Sinclair, *op. cit.* p. 67.

[7] Drummond Robinson, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 14, 'Vulval Discharges in Children.'

As regards the gonococcus itself, it is a diplococcus with a refractile envelope, generating by fission; it varies much in size, but neither form nor size[3] are reliable guides in its recognition; ill-developed gonococci may be sometimes found. Generally gonococci are found lying free, either in colonies or as separate and distinct diplococci, never forming chains; they may surround the pus cell, or lie on its surface, but the greater number are actually contained in the pus cell, and, both in this and in epithelial cells, cause cell destruction. Fuchsin is probably the best stain, while Gram's method is not necessarily a conclusive test.[4] Sometimes the gonococci are few in number, or may be mixed with other organisms, or be hidden in deeper portions of the mucosa, rendering their recognition difficult.

The chief points, besides these, in favour of the gonococcus being more or less the factor of the disease, are that gonococci exist in the pus of every untreated case at some period or other. Again, secretions free from gonococci do not cause a gonorrhœal inflammation, while secretions containing gonococci, even in small quantities, will affect mucous membranes with certainty; still one cannot say that if gonococci are not found the disease has not been gonorrhœal.[5] Cultivation and inoculation experiments tend perhaps still further to support the evidence that the gonococcus of Neisser is the cause of the disease we call gonorrhœa.

Results of Gonorrhœal Infection in the Female.—Generally speaking, gonorrhœa is seen in the acute or the chronic form.

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[1] McCann, *op. cit.* p. 236; *id. Encycl. Med.* vol. iv. p. 196.

[2] *Der Mikrorganismus der Gonorrhöischen Schleimhaut-Erkränkungen*, 1885, Wiesbaden.

[3] McCann, *op. cit.*; see also chapter on 'Micro-organisms in Relation to Diseases of Women' in this book.

[4] McCann, *op. cit.*

[5] McCann, *op. cit.*

In the *acute* there is generally some reddening and swelling of the vulva, with purulent discharge, and pain on micturition; but it is curious that in women[1] little notice is often taken of urethral troubles as compared with the male, and the most serious infection of the uterus and pelvic organs may take place without the patient having even noticed any discomfort with micturition. Sometimes much swelling of the vulva occurs, with great tenderness, and Bartholin's glands become infected, painful, and swollen; the inguinal glands may inflame. The urethra, in well marked cases, is thickened, swollen and tender, and pus can be squeezed from the orifice.

Much discussion has arisen on the subject of the occurrence of a true gonorrhœal vaginitis, and the author agrees with Sinclair that it is an uncommon occurrence. The vagina seems to be the last portion of the genital tract from the uterus downwards to be affected.[2] Urethral and uterine gonorrhœa may be well marked in some cases without any definite changes in the vagina. Probably[3] the squamous epithelium of the vagina, which is very like true skin, long resists infection or the processes of disease; still, in children and young females the vaginal lining is softer than in adults, while the softening and increased vascularity of pregnancy may render the surfaces of the vagina less resistant.

McCann[4] states that 'practical experience teaches us that we do meet with cases of gonorrhœal vaginitis.' While admitting the arguments, the author thinks its occurrence is certainly rare.

With regard to the infection of the uterus, such infection is the rule, not the exception,[5] especially that of the glands and mucous membranes of the cervix, the latter being swollen and reddened, and there is acute or chronic catarrh, accompanied with erosion; in some cases pus may be seen pouring from such a cervix.

The disease produces an endometritis, and though endometritis is more often caused by septic puerperal infection, the poison of gonorrhœa is certainly one of the factors of its causation, and the process may extend to the Fallopian tubes, ovaries, and peritoneum.

The Fallopian tubes are affected in acute gonorrhœa with-

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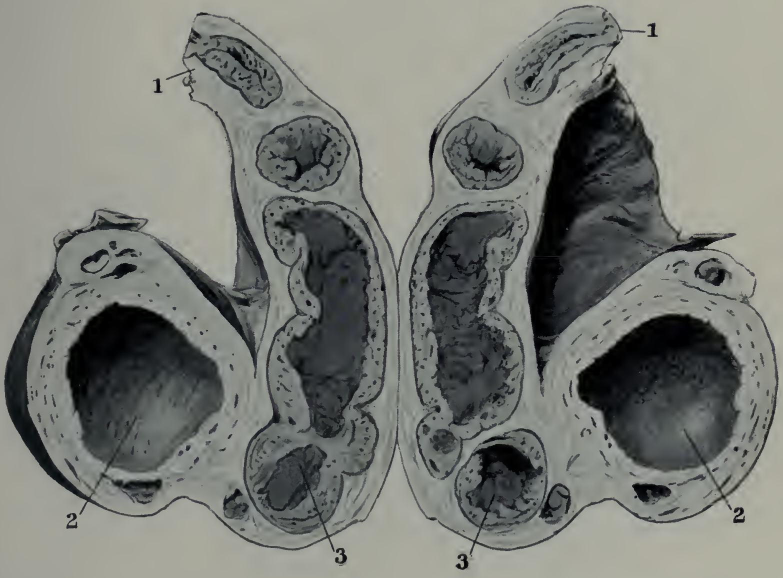
[1] Sinclair, *Gonorrhœal Infection in Women*, p. 81.
[2] Sinclair, *op. cit.* p. 82.
[3] Bumm, *op. cit.*
[4] McCann, *op. cit.* p. 245.
[5] Sinclair, *op. cit.* p. 84.

Fig. 10.

PURULENT SALPINGITIS, WITH ABSCESS OF THE OVARY (GONORRHŒAL).



A Fallopian tube and ovary which have been divided by longitudinal section. The tube was distended with pus. It is very tortuous, so that the lumen is only exposed in segments. The walls are very thick, and the mucous membrane swollen. In the ovary there is a granulating cavity, the sac of an abscess which probably originated in a cyst of the ovary; it does not communicate with the tube.

1, 1. Uterine end of Fallopian tube.

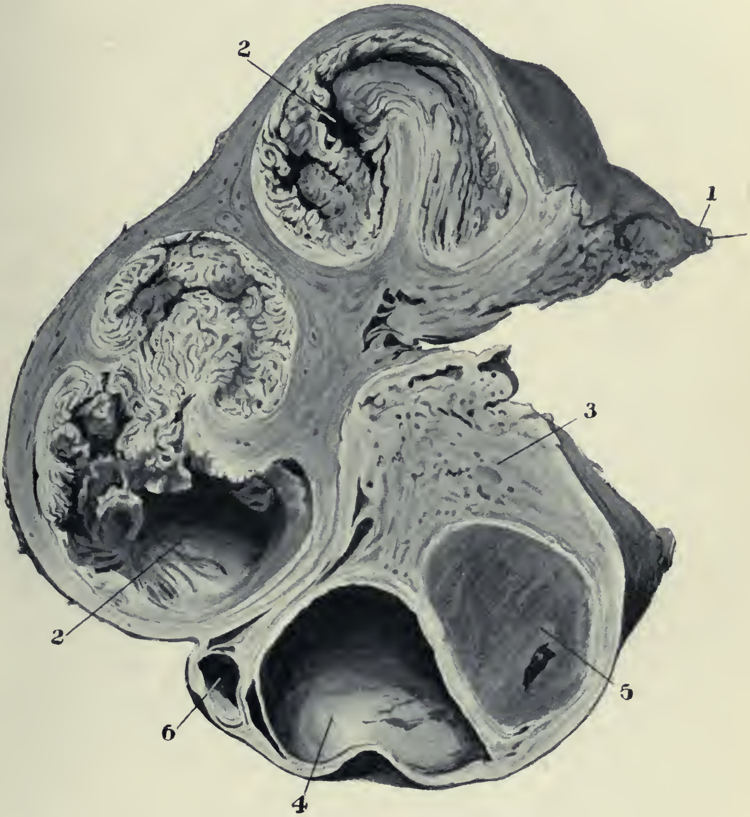
2, 2. Abscess in the ovary.

3, 3. Portion of lumen of the inflamed tube; the mucous membrane is much inflamed and thickened.

(Specimen 2386. Museum, St. Thomas's Hospital. See Cullingworth, 'Clinical Illustrations of Diseases of the Fallopian Tube,' plate v. fig. 2.)

Fig. 11.

SUPPURATING FALLOPIAN TUBE (PYOSALPINX) AND OVARY DUE TO GONORRHŒA.



1. Uterine end of Fallopian tube, into which a bristle is passed.

2, 2. Large suppurating loculi; the mucous membrane is swollen and voluminous, and coloured yellow from infiltration of pus. The walls of the tube are much thickened.

3. The ovary, which is enlarged and contains three cysts (4, 5, 6).

4. Cystic cavity in the ovary which contained pus (ovarian abscess).

5. Large cyst filled with recently extravasated blood.

6. A small blood-cyst.

(Specimen 2388. Museum, St. Thomas's Hospital. See also Cullingworth, 'Clinical Illustrations of Diseases of the Fallopian Tubes,' plate iii. fig. 1.)

out doubt, but we know little of the changes that occur; but in the more chronic cases similar changes occur to those described in Diseases of the Fallopian Tubes (q.v.) whereby the fimbriated extremities are closed, leading to dilatation and the formation of hydro- and pyosalpinx. More will be said of this in speaking of the chronic cases.

Extension may take place in acute cases to the peritoneum, but a general gonorrhœal peritonitis must be very rare. Generally the bowels and omentum are matted together by adhesions, as in ordinary pelvic inflammation, forming a barrier against a further extension of the process.

In its *chronic* forms, gonorrhœa may show itself in the general ill-health, persistent discharge, painful menstrual periods and pelvic distress, or even a definite attack of pelvic peritonitis, with its subsequent sterility.[1] Such cases are but too often seen in young women a year or less after marriage. In such the orifice of Bartholin's glands may be found inflamed, the cervix swollen, and its mucosa red and accompanied with erosion (adenoma); movement of the uterus causes pain, owing to tenderness of the surrounding organs; the ovaries are frequently enlarged, tender, and prolapsed, or they may be fixed by surrounding perimetritis.

The changes in the Fallopian tubes [2] in chronic gonorrhœal infection are chiefly matting of the fimbriæ and closure of the abdominal ostium, and the subsequent formation of hydro- and pyosalpinx. The ovary shares in the general matting and adhesion of the pelvic viscera, perioöphoritis occurs, but it is but a part[3] of the general inflammatory process (perimetritis). Generally, tube and ovary are closely bound together by dense adhesions; sometimes such ovaries are filled with small cysts or abscess cavities, which may form a single pus cavity (ovarian abscess, figs 10, 11), in other cases the pyosalpinx and the ovarian abscess open into one another.

It is rare for such abscesses to burst into the general peritoneal cavity, but a few fatal cases [4] have occurred. They appeared to be due to definite destructive ulcerations of the tube, even though not distended.

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[1] Sinclair, *op. cit.* p. 93.

[2] Lewers, 'On the Frequency of Pathological Conditions of the Fallopian Tubes,' *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 199.

[3] Olshausen, *Die Krankheiten der Ovarien*, Stuttgart, 1877.

[4] Cullingworth, 'Etiological Importance of Gonorrhœa,' *Brit. Med. Journ.* July 20, 1899, case II.; Lewers, *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 298; Grigg, *Journ. Brit. Gyn. Soc.* vol. ii. p. 264.

It is not to be supposed that all cases of gonorrhœal salpingitis run to abscess or pyosalpinx. Many cases get well, and the attack runs the course of an ordinary perimetritis.

The most serious result perhaps is sterility,[1] owing to the closure of the abdominal ostium of the tubes. Generally such sterility is relative; patients frequently have had one child. The same holds good of the sterility of prostitutes.

It is of course possible that the infection of gonorrhœa may take place during pregnancy, or even shortly after delivery; such cases may be severe in their results, and Sänger[2] professes to recognise a gonorrhœal puerperal fever. Sinclair and Noeggerath think that such cases recover slowly, because of the pelvic adhesions, whereas women who survive a genuine attack of puerperal sepsis soon get well.

Another point in relation to this subject is the infection of the conjunctivæ of newborn infants, but not all forms of ophthalmia neonatorum are necessarily gonorrhœal. Stephenson,[3] however, found gonococci in sixty-six per cent. of his cases.

Gonorrhœal rheumatism in women also occurs as a complication, with definite effusions into joints, such as the wrists or knees, and ankylosis may even occur.

As to the character of the infection causing the complications of gonorrhœa, it is possible, as McCann states, that we may have—[4]

a. Pure gonorrhœal infection, the result of the infection of gonococci alone.

b. Mixed infection, the result of the invasion of pus organisms which have developed in the mucous surface originally attacked by the gonococci.

c. Secondary infection, where the gonococci produce the complication followed later by the entrance of pus organisms, both existing together until the gonococci die or are overgrown by the pus cocci.

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[1] Matthews Duncan, *Gulstonian Lectures*, 1883, and W. J. Sinclair, *op. cit.* p. 119.

[2] Sinclair, *op. cit.* pp. 125, 127.

[3] Stephenson, *Encycl. Med.*, vol. ii., note on p. 354.

[4] McCann, *op. cit.* vol. iv. pp. 194 and 195.

d. Toxic infection, where the complications may be due to the result of the absorption of toxic products produced by the gonococci or the pus organisms.

With regard to latent gonorrhœa, Noeggerath believes that a man is never completely cured, and that, if married, he is certain to affect his wife; such cases are better termed chronic, not latent.

Gonorrhœa can, of course, be cured completely in either sex by appropriate and early treatment, but the eradication of the infection from the glands of the cervix is a most difficult matter.

CHAPTER V

ON MENSTRUATION AND ITS DISORDERS

Menstruation is the expulsion of the broken-up endometrium and blood from the uterus at the monthly times, or at least this is the ordinary explanation, but the precise nature of this phenomenon is still unknown and still debatable.

The average age of the first menstruation [1] in this country is fourteen, and the cessation about forty-five; the average rhythm is about twenty-eight days, counting from the beginning of one period to the beginning of the next. It recurs every three weeks, or perhaps it may be better put as lasting five days on the average, the woman being 'clear' three weeks. The quantity lost varies greatly; generally the amount is four to six ounces. Its colour is prune-juice, it is acid and does not normally coagulate, and is probably prevented from so doing owing to the admixture of the secretion from the cervical and uterine glands. Its coagulation in cases of profuse menstruation is due to the excess of blood over the mucus. The reaction is due to the vaginal acidity (see chapter on Diseases of the Vagina). Under the microscope menstrual blood shows red and white corpuscles, corpuscles in a state of fatty degeneration, and uterine, cervical, and vaginal epithelium.

Changes in the Uterus.—Menstruation is the evidence of changes in the uterus itself. The mucosa, as pointed out by Williams,[2] really consists of the whole thickness of the uterine wall to the peritoneum, the muscle of the uterus being really an enormous muscularis mucosæ, for the human fœtal uterus shows a distinct submucous layer just beneath the peritoneum. The layer known as mucous membrane is the part internal to the muscular coat. On the average it is $\frac{1}{25}$th inch thick; at the height of its growth just before each menstrual period, it may be $\frac{1}{8}$–$\frac{1}{4}$ inch thick, but after the first menstruation it never returns to its original thinness, but remains in the intervals about $\frac{1}{12}$ inch thick.[1]

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[1] Champneys, *Painful Menstruation*, London, 1891, p. 2; Harveian Lectures, 1890.

[2] Sir J. Williams, *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 112.

The most discordant views are held with regard to the changes in the endometrium, and they are fully discussed in Champneys' Harveian Lectures on 'Painful Menstruation' for 1890, since published in book form, to which the writer would refer those interested. Champneys also gives the literature of this enormous subject.

Much of the difficulty arises from the trouble of obtaining material for observation, as healthy uteri are not removed during life, and diseased uteri are not suitable for observation, and mere curettings are of little value.

Champneys quotes the observations of Kundrat and Engelmann, Sir J. Williams, Leopold, Wyder, de Sinéty, Möricke, and Minot, and the facts observed and the theories upheld are somewhat contradictory: fatty degeneration of the mucous membrane, the nature of its increase in thickness, the cause of its decrease in thickness, and the cause and nature of the bleeding are still matters of dispute.[2]

Most observers agree that before menstruation the human endometrium is thicker than after it has occurred, and that it grows again in the intermenstrual period, but varied views are held on the subject of disintegration, and if so, how much is disintegrated and if there be a so-called 'raw surface' left, on which, should pregnancy ensue, an ovum would engraft itself. Taylor[3] holds that probably this does not occur at all, and that the ovum tends far more to 'settle' and get embedded on the full-grown mucosa of the ante-menstrual period, and that in questions of conception the onset of the menstrual flow means that the chance of pregnancy for the time being is 'lost,' and it is very questionable whether the epithelium is removed to any extent by menstruation. In observations on animals Heape[4] comes to the conclusion that shortly after menstruation there is growth of the interglandular tissues, vessels increase, and there are areas of congestion and extravasation, which gradually blend, forming spaces, these fill with blood, and finally rupture into the uterine cavity, destroying some at least of the endometrium, which is regenerated by the embryonic tissue of which it is composed.

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[1] Champneys, *op. cit.* p. 6.

[2] Champneys, *op. cit.* p. 10.

[3] Taylor, *Extra-Uterine Pregnancy*, p. 13.

[4] Heape, 'Menstruation in Monkeys,' *Phil. Trans.* vol. clxxxv. 1894, p. 411, and 'Menstruation and Ovulation of Monkeys and the Human Female,' *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 161.

The interest of these observations coincides somewhat with Eden's researches[1] on the Placenta, especially in the hæmorrhages which occur in the superficial or compact layers of the decidua serotina, due to the rupture of delicate vessels, forming thrombosed areas into which the chorionic villi find their way. These spaces are the first indications of the future placental intervillous spaces.

With reference to Heape's observations on monkeys the question is, can such be compared to the human female? Hæmorrhage certainly seems to be a factor in all observations, but whether actual disintegration occurs is disputed. Herman[2] thinks that such breaking up and subsequent degeneration may be fairly assumed to occur.

A factor less disputed in menstruation is the occurrence of uterine contractions. These certainly occur in pregnancy, and with fibroids during menstruation,[3] and may cause regurgitation after opening a hæmatocolpos.[4]

Herman[5] points out the fact that the cervix dilates during menstruation.

Condition of the Pelvic Organs during Menstruation.—There is an increased supply of blood to the pelvic organs and the vascular tension is greatest on the day before the flow comes on.[6]

Observations on fibroids prove their increase in size before the flow, and their subsequent diminution after it is once established. Piles are worse at this period, the cervix and vagina are more vascular, and there is a feeling of fulness with aching in the pelvis.

Champneys sums up the question thus[7]: 'I believe that the thickening of the mucous membrane is due to growth; the bleeding is due to increased afflux of blood to the uterus in common with the pelvic organs, on the top of which comes uterine contraction, which determines the hæmorrhage.'

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[1] Eden, *Journal of Path. and Bacteriology*, Jan. and. Dec. 1896.

[2] Herman, *Diseases of Women*, p. 516.

[3] Sir J. Williams, *Lancet*, vol. i. 1880, pp. 764, 873.

[4] Champneys, *op. cit.* p. 13.

[5] Herman, 'On the Change in the Cervical Canal during Menstruation,' *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 250.

[6] Giles, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 115.

[7] Champneys, *op. cit.* p. 15.

The relation between ovulation[1] (which is the liberation of the ovum from the ovary) and menstruation (which is the monthly preparation of the uterus for the fertilised ovum) is, that ovulation does not invariably coincide with menstruation, though it generally does,[2] since conception may take place at any time in the human female. Menstruation depends on ovulation, for if the ovaries are imperfect menstruation is absent, or if removed menstruation stops, and menstruation, ovulation and conception end at the climacteric.

Dysmenorrhœa.—A woman who suffers pain in connection with menstruation is said to be the subject of dysmenorrhœa; but not all pains are dysmenorrhœal. The pain must be limited to the pelvic organs or in the genital sphere;[3] this is limited by the iliac crests above, and the knees below. Again, not all women bear pain alike, and the personal equation of pain in any one individual is difficult, if not impossible, to estimate.

Classification.—The only real dysmenorrhœa is the Spasmodic variety, with its subdivision, Membranous dysmenorrhœa.

Spasmodic dysmenorrhœa is the neuralgic, mechanical, or obstructive dysmenorrhœa of other writers.

Other varieties of dysmenorrhœa are described; they are not, as such, dysmenorrhœa at all;[4] they are pain associated with such diseases as—

a. Chlorosis and Anæmia.
b. Gout and Rheumatism.
c. Fibroids.
d. Inflamed ovaries.
e. Diseases of the tubes.
f. Congestion and inflammation of the pelvic organs.

Hence it is not the mere dysmenorrhœa which is the factor, but the disease and the pathological product on which such pain depends, and to make a separate classification of dysmenorrhœa for each such pathological product is illogical and unnecessary.

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[1] Herman, *Diseases of Women*, p. 518.

[2] Heape, *op. cit.*

[3] Champneys, 'Pain in Pelvic Cancer,' *Trans. Obstet. Soc. Lond.* vol. xxii. 1880, p. 5, and *id. Painful Menstruation*, p. 43.

[4] Champneys, *Painful Menstruation*, p. 19.

Many anæmic girls suffer from all varieties of pain; some, indeed, suffer from true spasmodic dysmenorrhœa, but there is no special dysmenorrhœa peculiar to anæmia.

Nor is there any such in connection with gout and rheumatism. With fibroids, there is often pain, pain from pressure, or pain from uterine contractions. Fibroid uteri swell during the intermenstrual period, and just before its onset, not with the onset. The woman suffers most *before* the flow comes on; as soon as it occurs the pelvic congestion is relieved, and she is more or less comfortable. Dysmenorrhœa is pain *with* the periods, not in the interval; here, again, it is the fibroid, not the mere pain, which calls for attention.

Ovarian dysmenorrhœa, dysmenorrhœa due to diseased tubes, and congestive dysmenorrhœa, are all inflammatory: that is, the pain is due to pelvic inflammation or its results at some time or other. There may be adhesions and peritoneal thickening, and the return of venous blood is impeded; there is fulness, aching, and pain, not at the menstrual period, but before it, and the flow is a safety-valve for the pelvic congestion which causes the pain. Such pain is again intermenstrual; the suffering is inversely proportionate to the amount of the flow.[1] Constipation is often a great factor of pain in these inflammatory cases.

Some attention has lately been drawn to the condition called 'Mittelschmerz,' by Addinsell[2] and various German observers, but very little is known, and it is doubtful if it can be recognised as a definite disease.

Membranous Dysmenorrhœa.—This is painful menstruation associated with the discharge of membranes from the uterus, accompanied by sensations which are unmistakable, and if the membrane be passed in more than one piece the sensations are repeated. The frequency[3] of the occurrence of membranous dysmenorrhœa of this sort is hard to determine, but it occurs oftener than is supposed, and it is known that some women pass membranes without pain.

The etiology of membranous dysmenorrhœa is doubtful; the disease is probably inflammatory, and inflammatory causes may conceivably be invoked, for a large number of cases follow pelvic inflammatory attacks, also abortion and parturition.[1] Much has been said of the excess of inflammatory fibrous tissue both in the ovaries and uterus as a cause of this condition.[2] Champneys takes the view that such cases are not the cause but the effect of the long-continued irritation from membranous dysmenorrhœa, for these changes may occur late in such cases.

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[1] Champneys, *op. cit.* p. 22.

[2] Addinsell, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 3.

[3] Sir J. Williams, *Trans. Obstet. Soc. Lond.* vol. xxiv. 1882, p. 117, and Champneys, *op. cit* p. 36.

Pathology.[3]—A dysmenorrhœal membrane is a triangular cast of the uterus about $1\frac{3}{4}$ in. long, 1 in. broad, and $\frac{1}{25}$–$\frac{1}{12}$ in. thick; it may be discharged whole, or in two or more pieces; the outer surface is rough and shaggy, and the villous processes on this surface may be $\frac{1}{4}$ in. long. Its inner surface is smooth, but marked with sulci and pits, which are the openings of the glands. The sac has three openings: one large for the cervix below, and two smaller above, for the Fallopian tubes. The substance of the cast[4] is connective tissue and many round cells; vessels and glands occur in varying amount, the glands being lined with columnar epithelium, which covers the inner surface of the sac. The shaggy prolongations on the outer surface are the processes of the membrane, not glands. The characteristic of the condition is cell proliferation, unlike the ordinary condition of uterine mucous membrane, but resembling that at the beginning of pregnancy (Champneys).

Leopold, Wyder, and others, do not regard the disease as one by itself, but a complication of such conditions as chronic metritis, flexions, fibroids, &c., *i.e.* inflammatory; but membranous dysmenorrhœa may certainly be primary, *i.e.* occurring with the first menstruation, and in many of Champneys' cases none of the above conditions were found.[5] Wyder describes large cells, only seen in the decidua of pregnancy, which might serve to distinguish real membranous dysmenorrhœa from early abortions; but mere decidual cells mean nothing, as they are only altered connective-tissue cells of the uterus.

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[1] Champneys, *op. cit.* p. 35.

[2] Sir J. Williams 'On the Natural History of Dysmenorrhœa,' *Trans. Obstet. Soc. Lond.* vol. xxiv. 1882, p. 125.

[3] Sir J. Williams, 'Pathology and Treatment of Membranous Dysmenorrhœa,' *Trans. Obstet. Soc. Lond.* vol. xix. 1877, p. 138.

[4] Champneys, *op. cit.* p. 38.

[5] *Op. cit.* p. 39.

The products of conception,[1] with which dysmenorrhœal casts might be confounded, are referred to in the chapter on Extra-uterine Gestation. Other things, such as fibrin, blood-clot, coagulated mucus, casts of the vagina, bladder, or uterus (as in cases of phosphorus poisoning and cholera), and foreign bodies, may be passed, and mistaken for dysmenorrhœal membranes (Champneys).

Finally, although membranous dysmenorrhœa frequently follows inflammatory attacks, abortion, parturition, and the growth of fibrous tumours, it is essentially a primary dysmenorrhœa, and in the majority of cases begins with the establishment of menstruation.[2]

In such cases the flow is profuse, or at least not scanty; pain is not relieved by the flow, but is relieved by the passage of the membranes; sterility is the rule, and the pain generally persists till the menopause. Membranous dysmenorrhœa sometimes runs in families.

The detachment of the membrane takes place from below upwards; the membrane is a foreign body; the cause of the pain is probably due to 'abnormal, partial, unconsentaneous' uterine contractions causing a colic, and the colic is super-excited by the passage of the membrane, which is a foreign body, over the specially sensitive internal os.[3]

That the internal os is the sensitive point in spasmodic dysmenorrhœa is proved by the passage of a sound in such cases.

Spasmodic Dysmenorrhœa.[4]—This is really a neurosis, and is often associated with other nervous phenomena. The pain is a colic; it may be very severe,[5] even agonising. It is an affection of an immature uterus; it precedes the flow, or it may occur without any flow at all. Its intensity varies with the abundance of the flow.

It is again essentially a primary dysmenorrhœa; the pain generally lasts twenty-four to forty-eight hours, and tends to get worse in time; sterility is the rule, but parturition cures the condition, just as may dilatation in some cases.

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[1] See also Eden, 'Spurious Abortion, with an account of three cases,' *Brit. Med. Journ.* Nov. 20, 1897.

[2] Champneys, *op. cit.* p. 42.

[3] Champneys, *op. cit.* pp. 46, 48.

[4] Matthews Duncan, *Clin. Lect.* 4th ed. p. 146.

[5] Herman, *Diseases of Women*, p. 60.

The **pathology** of the subject cannot be settled, as Champneys[1] says, on mere mechanical theories. Flexions of the uterus causing obstruction have never been seen, nor are flexions necessarily associated with dysmenorrhœa. Herman's[2] and Vedeler's[3] statistics are of the greatest importance in the matter. Pathological anteversion is the result of pelvic inflammation; here the pelvic inflammation is the cause of pain. Conical cervix and pin-hole os are not causes of dysmenorrhœa; they are evidences of mal-development, and such mal-development of the uterus is a common cause of dysmenorrhœa, but no pin-hole os has ever been seen that could not transmit 'one drop of blood in three minutes'; for, if four ounces is the average loss in menstruation during four days, this gives one ounce a day, twenty drops an hour, or one drop in three minutes[4] (Champneys).

The pain caused by fibroids has already been discussed.

Congestion[5] caused by flexion cannot be a cause of pain. Flexions either forwards or backwards do not and cannot cause congestion, owing to the circular distribution of the uterine vessels.[6] Even the tightest ligature round the uterus could cause no 'embarrassment' of the circulation except at the exact situation of the ligature.

Real Obstructive Dysmenorrhœa.—This can only occur with cicatricial contraction of the cervix as in cancer, or in atresia, following operation.

There may be absolute or relative atresia; the latter is only discovered on withdrawing the sound. In such cases menstrual blood may be retained and cause painful uterine contractions; a fibroid might possibly do the same thing.

The analogy is also seen in congenital atresia of the genital canal leading to Hæmatocolpos and Hæmatometra; the monthly sensations occur, although the menstrual blood is retained.

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[1] Champneys, *op. cit.* p. 60.

[2] 'On the Relation of Anteflexion to Dysmenorrhæa,' Herman, *Trans. Obstet. Soc. Lond.* vol. xxiii. 1881, p. 209; and on 'Relation of Backward Displacements to Dysmenorrhœa,' *ibid.* vol. xxiv. 1882, p. 161.

[3] Vedeler, *Arch. f. Gyn.* vol. xxi. p. 211. See Champneys, *op. cit.* p. 70.

[4] Champneys, *op. cit.* p. 64.

[5] Herman, *Lancet*, vol. i. 1881.

[6] Sir J. Williams, *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 112.

In Pyometra from cancer, great pain occurs if the uterus become distended.

Amenorrhœa.[1]—This term signifies absence of menstruation. Clinically, cases vary according to the following points :

- *a*. Menstruation may be suppressed after having been established : **suppressio mensium.**
- *b*. Menstruation may never appear, it stays away : **emansio mensium.**
- *c*. Menstruation occurs, but its products are retained : **retentio mensium.**

Suppressio Mensium.—In this form of amenorrhœa the menses are suppressed after having been established. Many of the causes of such amenorrhœa are physiological, and may exist as a normal condition in pregnancy, lactation, and the menopause. Others are pathological, and due to such constitutional conditions as anæmia, chlorosis, diabetes, morbus cordis[2] (in some cases), Bright's disease, malaria, cancerous cachexia, tuberculosis, acute illnesses and fever, pernicious anæmia, leukæmia and Hodgkin's disease. In fact, any disease which is associated with deficient nutrition or increased waste[3] is associated with anæmia, and such anæmia is the cause of amenorrhœa. It is not the object of the present work to discuss the causes of anæmia, for which other authorities may be consulted.

Much the same may be said of the wasting diseases causing amenorrhœa. All diseases that cause wasting at length stop menstruation, such amenorrhœa is conservative,[4] and the amenorrhœa does not come on till the disease is advanced. Such diseases are phthisis, diabetes, caries of bone, anorexia nervosa, and febrile illnesses. Gastric ulcer is not an uncommon cause both of anæmia and amenorrhœa.

Nervous diseases connected with suppression or even the non-appearance of menstruation are seen in the melancholic or associated with imbecility, cretinism, and insanity. Herman mentions exophthalmic goître as associated with amenorrhœa.

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[1] Matthews Duncan, *Clin. Lect.* 4th ed. p. 129.

[2] Gow, 'On Relation of Heart Disease to Menstruation,' *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 126. [Note.—In cases observed by the author, menorrhagia has been commoner with heart disease than amenorrhœa.]

[3] Herman, *Diseases of Women*, p. 558.

[4] Herman, *op. cit.* p. 563.

Some cases of amenorrhœa are associated with mental shock.

Apart from mere nervous conditions, some change in the mode of life may be accountable, as in girls who go from the country to city life, or to school in districts or countries away from home.

The physiological causes of amenorrhœa must be kept in mind, especially pregnancy and lactation. Serious mistakes may be made if they be forgotten, for conception may take place in the absence of menstruation.

Extreme youth or extreme old age are necessarily associated with amenorrhœa.

Another class of cases are those in which for some cause a premature climacteric ensues.[1] Some occur early in life and may be associated with puerperal atrophy or excessive involution of the uterus, or abscess or destruction of the ovaries,[2] or an artificial menopause may be brought about by the justifiable removal of both ovaries for the cure of disease. In such cases the cessation of the reproductive function is often followed by obesity, and by the nervous symptoms which follow the climacteric. These are of importance.[3]

Vicarious Menstruation.—This is merely mentioned with the subject of amenorrhœa since it has been thought that if the uterus does not menstruate a flow of blood from some other part may replace it. That such is possible is utterly absurd, and no such thing has ever been accurately observed or described.[4] That a loss of blood may occur is granted, but it is not menstruation; menstruation comes from the uterus and the uterus only. Histories given by patients of vicarious menstruation should be looked upon with great suspicion, nor can we trust those who reckon as menstruation the hæmorrhage of a disease which causes amenorrhœa, or state that they lose blood from the nose, stomach, bowel, or lungs, instead of menstruating. Sometimes such losses may even occur rhythmically each month, as if taking the place of menstruation, or they may occur in connection with the vascular tension which precedes menstruation; hæmorrhagic rashes may even occur and recur at these periods. In such cases, all that can be said is that hæmorrhage from other parts accompanies menstruation; it certainly does not replace it,[1] nor is it possible that any organ can usurp the functions of the uterine mucosa.

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[1] Champneys, 'On Removal of the Uterine Appendages,' *St. Bartholomew's Hosp. Rep.* 1893, p. 45.

[2] Herman, *Diseases of Women*, 'Super-involution,' p. 588; Targett, *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 216; and Keppler, *Arch. f. Gyn.* vol. xxxix. 1890.

[3] Tilt, *Change of Life*, 1882; Savage, *Med. Press and Circular*, 1893, and *id.*, *Insanity*, p. 73.

[4] Matthews Duncan, *Clin. Lect.* 4th ed. p. 135, says it is 'A tissue of error, if not of absurdity.'

Retentio Mensium.[2] (Figs. 12, 13.)—In this condition menstruation occurs, but its products are retained in consequence of atresia of the genital canal. The most common atresia is closure of the vagina by a transverse septum at its lower part. Such a septum used to be described as 'imperforate hymen,' but close observation will show that the atresia is above the hymen. Such atresia is due to a failure of the vagina, developed from the ducts of Müller, to reach and open into the deepened portion of the uro-genital sinus behind the vestibule.

The vagina may be closed, not simply by a transverse septum, but throughout the whole or part of its extent by connective tissue between the urethra and rectum, indicating a failure on the part of Müller's ducts to reach sufficiently far to complete the vaginal canal.

Retention of menses may be due not only to vaginal atresia, but to occlusion of any part of the genital tract from the os uteri internum to the vulva; thus, the whole of the lower part of the vagina may be occluded or absent, causing retention in the upper part of the vagina and in the uterus, or the seat of obstruction may be at the os externum or the os internum, giving rise to the retention of blood in the uterus[3] (**Hæmatometra**), the accumulation occupying the body and cervix in the one case, in the other the body alone.[4] In such cases the endometrium is atrophied and the muscular wall thin, and deposition of fibrin takes place.

Rarer and more complicated cases are those in which the whole or a portion of the utero-vaginal canal is divided into

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[1] Herman, *op. cit.* p. 612.

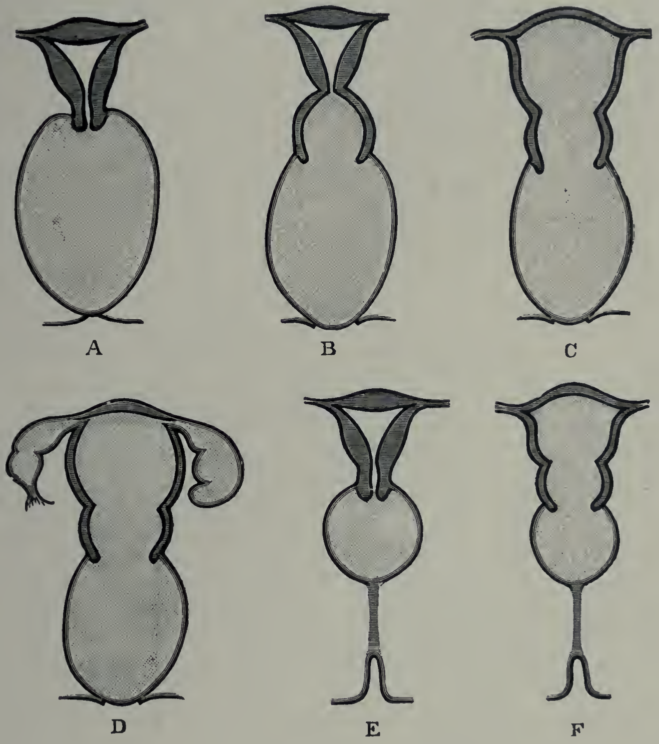
[2] Howard Kelly, *Operative Gynæcology*, vol. i. p. 549, 'The Uterus as a Retention Cyst,' and Herman, *op. cit.* p. 592; see also, *ibid.*, fig. 162, p. 593.

[3] Cullingworth, 'Retention of Menstrual Blood from Atresia of Vagina,' *Brit. Med. Journ.* Jan. 6, 13, 1900; *id. Med. Chronicle*, Oct. 1887; Lewers, *Diseases of Women*, 5th ed. p. 497.

[4] Cullingworth, Case V. *Brit. Med. Journ.* Jan. 13, 1900, p. 66.

Fig. 12.

DIAGRAMS ILLUSTRATING THE EFFECTS OF ATRESIA OF THE GENITAL PASSAGES.

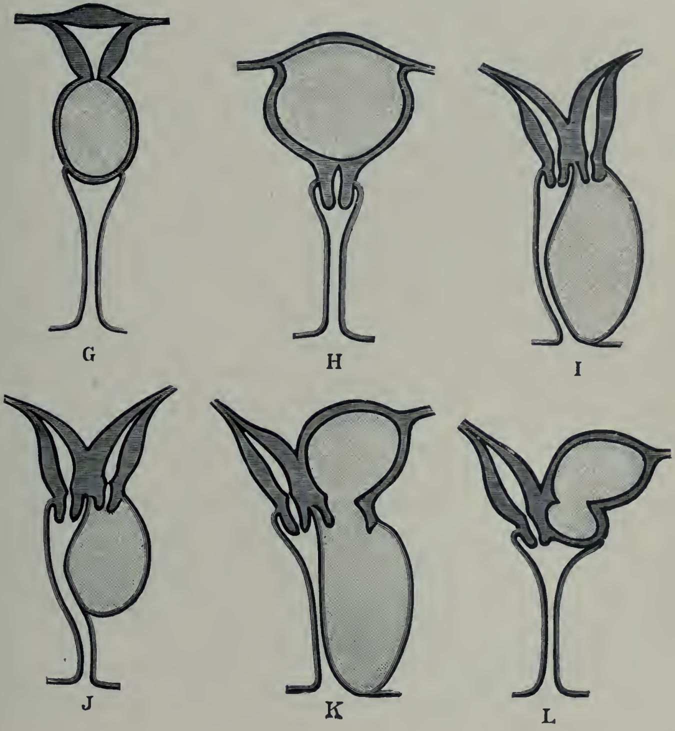


A. Hæmatocolpos (the atresia being at the vaginal orifice).
B. Hæmatotrachelon (cervix distended also).
C. Hæmatometra.
D. Hæmatocolpos, hæmatometra, and hæmatosalpinx.
E. Atresia of the lower or middle portion of the vagina, its upper part being distended.
F. Atresia of the lower or middle portion of the vagina, the vagina and uterus being distended; the lower portion of the vagina, if present (in E and F), is patulous.

(Bland-Sutton and Giles, 'Diseases of Women,' 1st ed. p. 69.)

Fig. 13.

DIAGRAMS REPRESENTING THE EFFECTS OF ATRESIA OF THE GENITAL PASSAGES.



G. Atresia of the os externum; the vagina is normal. Hæmatotrachelon first occurs; later, hæmatometra and hæmatosalpinx may follow.

H. Atresia of the os internum, pure hæmatometra resulting. This as a congenital condition is very rare.

I. Atresia affecting one-half of a double uterus or vagina (unilateral hæmatocolpos).

J. Unilateral hæmatocolpos (not reaching the vulva).

K. Unilateral hæmatocolpos and hæmatometra.

L. Unilateral hæmatometra (the atresia affecting the os externum).

In the diagram the various forms of atresia in cases of double uterus are represented as affecting a uterus bicornis, but similar changes are found in connection with uterus septus and uterus didelphys.

(Bland-Sutton and Giles, 1st ed. p. 70.)

two parts by a septum, and where one side is only occluded[1] (fig. 13, *i*, *j*, *k*, *l*,). Distension of the vagina by menstrual blood is called **Hæmatocolpos**, and if the unilateral occlusion be at the vaginal orifice, there is formed the puzzling condition known as Unilateral Hæmatocolpos;[2] if it be at the os uteri, the tumour is a **Unilateral Hæmatometra.**

Sometimes the Fallopian tubes are filled with blood. Such filling of the tubes with blood[3] is not mechanical, for in hæmato-salpinx associated with retained menses the tubes are not healthy, and their abdominal ostia have been closed by inflammation. This is said not to occur in the lower vaginal atresias due to congenital defect, but in the 'broad atresias,' as Herman calls them, which are not due to congenital defect, but are acquired and are a result of inflammation.[4] Such inflammation leads to destruction of the epithelium and adhesion of the opposed mucous surfaces, and the same inflammation spreads to the Fallopian tubes and closes their ostia. The pelvic congestion accompanying menstruation leads to bleeding in such diseased tubes, and the blood is now pent up. This only applies to the formation of hæmato-salpinx in connection with retained menses.[5]

Another curious fact about the Fallopian tubes in this condition is, the possibility of so-called regurgitation of blood from a hæmatometra into the peritoneal cavity. Two explanations are possible: either that one of the occluded tubes bursts[6] at the time of operation on the hæmatometra, or, owing to altered pressure relations[7] after the main accumulation of blood in the uterus has been set free, uterine contractions have forced some of the blood through the tubes into the peritoneal cavity.

The conditions which lead to a suspicion of retained menses are the occurrence of menstrual molimina in a woman who has never menstruated, and the presence of a swelling in the lower part of the abdomen; hence a local examination is imperative. Sometimes there is a bulging at the vulval orifice, but it is not common; most of these swellings due to retained menses result in enormous distension and thinning of the vagina, the uterus taking little or no part, being perched on the top of the hæmatocolpos,[1] or at least only the lower end of the cervix is expanded. The bladder is elongated and drawn up to the umbilicus along with the uterus, and compressed, sometimes causing retention of urine, while the rectum is encroached upon.

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[1] Bland-Sutton and Giles, *Diseases of Women*, pp. 69–70, 1st ed.

[2] Cullingworth, *op. cit.* p. 67; *Trans. Amer. Gyn. Soc.* vol. xviii. 1893, p. 434, On Unilateral Hæmatometra.'

[3] Herman, *Diseases of Women*, p. 595.

[4] Herman, *op. cit.* p. 596.

[5] A very large number of cases of Hæmatosalpinx are due to Tubal Pregnancy. (Author.)

[6] Fuld, *Arch. für Gyn.* vol. xxxiv., quoted by Herman, p. 597.

[7] Cullingworth, *Brit. Med. Journ.* 1900, vol. i. p. 7.

The characters of retained menstrual fluid are remarkable, consisting of thick, viscid, dark-brown fluid, of the consistence of treacle, which never clots. Sometimes it is tar-like; it contains much mucus. The quantity varies greatly, forty or even fifty ounces being reached. Matthews Duncan [2] quotes one of his own cases in which he measured more than fifty ounces.

In cases which are not operated on, rupture may take place through the obstructing septum, or into the rectum, bladder, cellular tissue, or through the perineum.[3] Rupture of a distended tube may set up fatal peritonitis. As long as the blood is shut up in the vagina and is not infected[4] by pathogenic organisms from the intestinal canal, it undergoes no change, but on opening such a cavity the conditions alter; some of the thick blood may not escape, and as it is dead tissue, it is the best possible medium for the development of putrefaction Hence great care is taken, as in all other operations, with regard to asepsis; douches and washing out the cavity afterwards are extremely dangerous for the same reasons.

The risks from rupture of the tubes or regurgitation have been explained.

Subsequent atresias may occur, after opening such a distended vagina; they are most difficult to treat. If the atresia be high up, much tissue has to be divided even to reach the accumulation, and the scar tissue rapidly contracts.

Atresia apart from congenital defects (fig. 14) may occur as the result of inflammation or cicatrisation after any kind of ulceration, or blocking of the cervix by a tumour or cancer, or after amputation of the cervix. If these occur during the

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[1] See chapter on Diseases of the Vagina, and fig. 4.

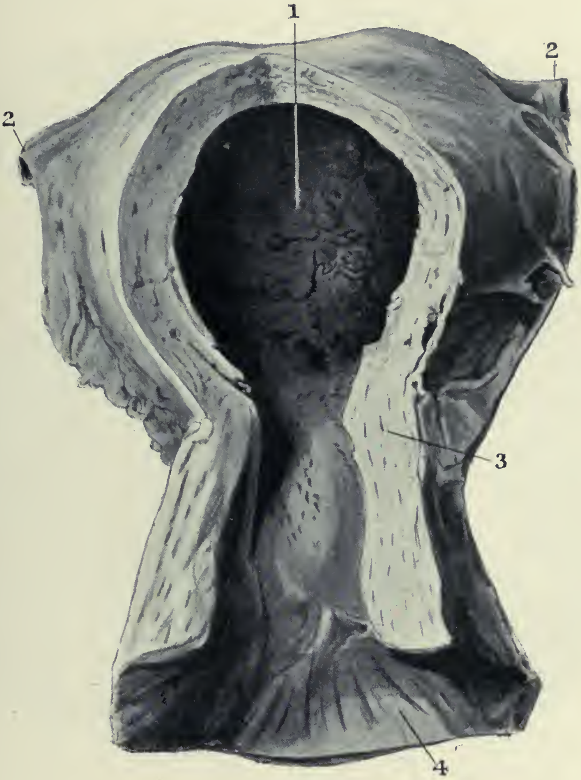
[2] Matthews Duncan, *Clin. Lect.* 4th ed. p. 109.

[3] Herman, *Diseases of Women*, p. 597.

[4] If infected, 'pyocolpos,' 'pyometra,' 'pyosalpinx,' may result.

FIG. 14.

HÆMATOMETRA.



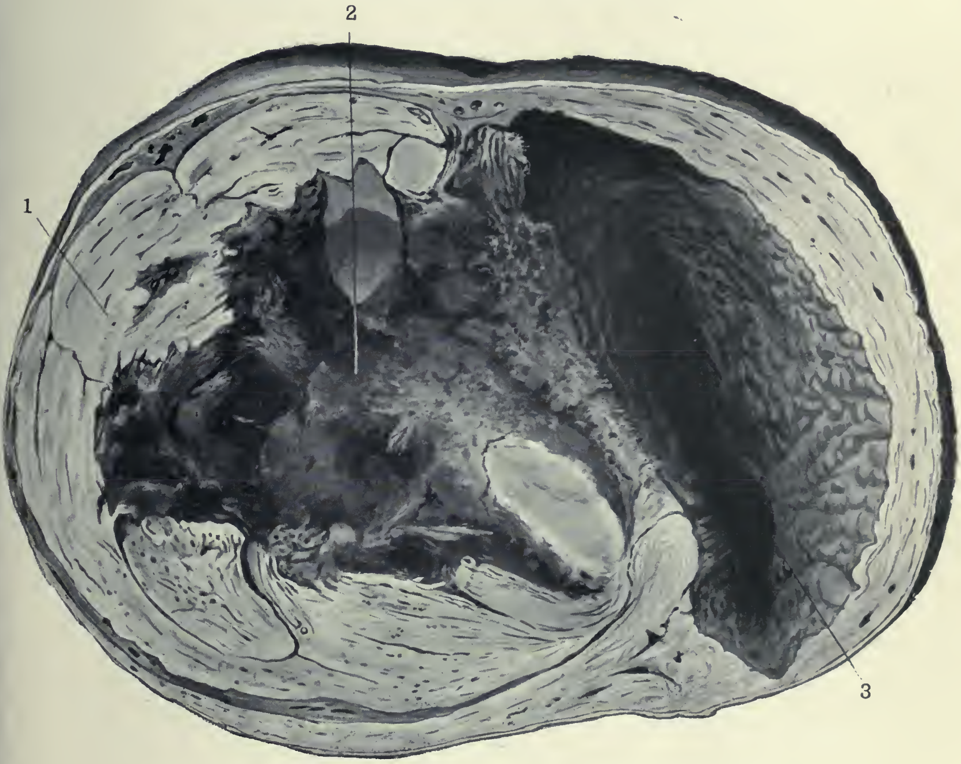
A uterus from an old woman of 60, which contained an ounce of broken-down dark blood-clot.

1. The much-distended body of the uterus, which was occupied by retained blood; it has stained the lining membrane a dark-red colour. No evident cause of the retention (such as cancer) could be found.	2, 2. Fallopian tubes. 3. Region of internal os. No obstruction, however, existed. 4. Vagina.

(Specimen 2965 B. Museum, St. Bartholomew's Hospital.)

Fig. 15.

HÆMATOMETRA CAUSED BY DEGENERATION OF A UTERINE FIBROID.



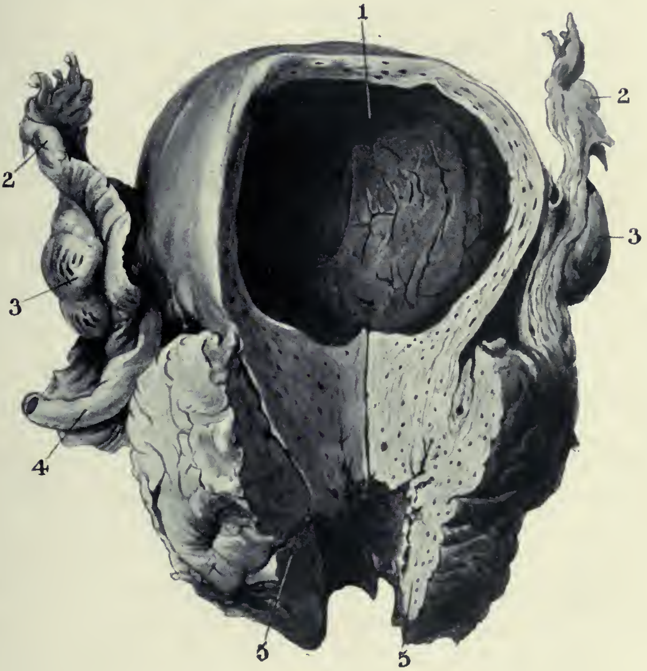
The tumour weighed 15 lbs., and was filled with retained blood of the consistence of mortar. A large degenerating fibroid is seen opening into the uterine cavity.

1. Healthier portions of the fibroid.
2. Cavity of the tumour which is degenerating, and which was filled with putty-like blood.
3. Enlarged cavity of the uterus, which was filled with blood, into which the fibroid opens.

(Specimen 4660 A; Museum, Royal College of Surgeons. The other half is 2956 A; Museum, St. Bartholomew's Hospital. See W. A. Meredith, 'Trans. Obstet. Soc. Lond.' vol. xxix. 1887, p. 422.)

Fig. 16.

PYOMETRA AND CANCER OF THE CERVIX.



A uterus, the cavity of which is considerably distended and which contained pus. Below, the cervix is everywhere involved by a cancerous growth blocking the canal.

1. Cavity of body of uterus, which contained pus.
2, 2. Fallopian tubes.
3, 3. Ovaries.
4. Round ligament.
5, 5. Large mass of cancer springing from the cervix.

(Specimen 2955. Museum, St. Bartholomew's Hospital.)

menstrual life of a patient it may lead to hæmatometra, often with great pain. In older women past the climacteric, especially with cancer, pyometra may result.

Meredith's case of Hæmatometra[1] occurred in a single woman of forty-six (fig. 15). She had a very large uterine fibroid which weighed over fifteen pounds; it measured twelve inches in height and twenty inches in breadth, and was composed of two distinct portions, limited externally by the line of attachment of the broad ligaments. The anterior portion consisted of a large interstitial fibroid, which constituted nearly two-thirds of the entire tumour. The remaining portion, formed by the posterior uterine wall, resembled in appearance a pregnant uterus; the tubes were thickened, dilated and closed; the left was filled with thick grumous blood. On a section of the tumour being made, the anterior portion consisted of a red fleshy fibroid with a calcareous capsule and a large central ragged cavity due to disintegration; this cavity communicated with the uterus by a large oval aperture measuring five by three and a half inches; both were filled with putty-like old blood.[2] The uterine cavity measured ten inches in length by four in breadth. The blood amounted in weight to about five pounds. The cervix was closed probably by some inflammatory atresia, and the enclosed blood gave rise to irregular recurrence of severe spasmodic abdominal pain, as in some of the cases previously described of hæmatometra due to congenital atresia.

Pyometra.[3]—This, in the vast majority of recorded cases, is due to cancer blocking the canal (fig. 16), or possibly to adhesions in endocervicitis. Sometimes with fibroids, the uterus contains a considerable amount of watery mucoid fluid and if decomposition occur, or the tumour be in a state of necrosis, such fluid is converted into pus.

In many of the observed cases, especially in old women with cancer of the cervix, the distension of the uterus (which is sometimes elastic) by pus[1] was accompanied with very great pain (M. Duncan). Kelly reports a case of this sort in an old lady of sixty-seven; the uterus contained six ounces of fœtid pus, and was as large as a two-and-a-half months' pregnancy.

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[1] Meredith, *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 422; St. Bartholomew's Hospital Museum, Specimen 2956A.

[2] Meredith, *op. cit.* p. 425.

[3] W. Tate, 'Three Cases of Pyometra complicating Cancer of the Cervix,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 323; H. Kelly, *op. cit.* vol. i. p. 552; M. Duncan, *Trans. Obstet. Soc. Lond.* vol. xxi. 1879, p. 54; Galabin, *ibid.* vol. xxii. 1880, p. 239; Griffith, *ibid.* vol. xxix. 1887, p. 398; Specimen, St. Bartholomew's Hospital Museum, 2955.

In other cases it does not appear necessary that there should be any obstruction at the cervix. In Galabin's[2] case there was no obstruction to the cervical canal, the uterus measured three inches in diameter, and there were cancerous changes near the internal os.

Griffith's[3] case was due to the obstruction of the uterine canal by a fibroid, the upper part of the uterus being converted into a pyometra.

Other cases of pyometra[4] have been recorded in which the condition was associated with imperforate hymen, or followed cicatrisation of the cervix or vagina from injury during childbirth or from ulceration of a non-malignant character; others occurred in connection with uterus bicornis and uterus septus. (See Hæmatometra etc.).

In nearly all observed cases of pyometra with cancer, the pus has been noted as extremely fœtid. The temperature may or may not be raised.

According to Bürkle's statistics quoted by Sinclair and Tate, pyometra occurs with cancer of the cervix in about 6·2 per cent. of cases. Tate's[5] figures show it as occurring in 11 per cent. of his cases. A large number occur in very old women.

Physometra.—This is a term used to designate a collection of gas in the uterus. It is usually found in pregnancy or in the puerperal condition, and associated with sepsis. Kelly[6] says that in gynæcological cases physometra is a rare accompaniment of a pyometra, and is probably due to the presence of gas-producing bacilli. He reports three cases: two complicating large, sloughing, submucous fibroids, and one associated with a pyometra due to cancer of the cervix.

In such cases the uterus may be markedly distended and tympanitic, and contain horribly fœtid gas and fluid.

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[1] Lewers, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 14.

[2] Galabin, *op. cit.* p. 239.

[3] Griffith, *op. cit.* p. 398.

[4] W. Tate, *op. cit.* p. 329.

[5] W. Tate, *op. cit.* p. 324.

[6] H. Kelly, *Operative Gynæcology*, vol. i. p. 553.

The cause of such tympanites in the uterus is said to be due to the Bacillus aërogenes capsulatus. Kelly[1] notes that the most extensive gas formations in the tissues of the mother were found after death. Whether the bacillus capsulatus can produce gas in healthy tissues the writer believes has not yet been satisfactorily proved, the nature of the infection being more probably terminal.[2]

Emansio Mensium.—In a few cases menstruation may be delayed very late, as the disease which causes the amenorrhœa may still exist, but in the great majority of true cases of absence of menstruation there is imperfect development of the uterus or ovaries or both. Yet deficient development of the uterus may occur with no defect of the ovaries, and it must be remembered that all uterine defects do not necessarily cause amenorrhœa, but only those that are associated with defects of growth.

Absence of the ovaries is very rare, and reliable cases are few; sometimes only one ovary can be detected, but this does not necessarily entail either amenorrhœa or sterility. A rudimentary condition of the ovaries[3] is more common; it is generally bilateral, and the ovaries are small and elongated as in the fœtal condition. In such cases the uterus is frequently imperfectly developed, and all evidences of the sexual functions may be absent; the breasts are small or absent; there is absence of pubic hair and deformity of the external genitals; the pelvis may remain in the infantile or child-like condition; menstruation and conception never occur.

Deformities of the uterus are described later. Those producing amenorrhœa are absence, rudimentary condition, or where only one half is developed. Menstruation in a double uterus is as perfect as in a normal one. Female pseudo-hermaphrodites are generally sterile.[4]

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[1] Dobbin, quoted by Kelly, *Johns Hopkins Hospital Bulletin*, Feb. 1897, and H. Kelly, *op. cit.* vol. i. p. 555.

[2] *I.e.*, the infection only occurs during the last hours of life. Post-mortem emphysema may be due to this bacillus; see chapter on 'Micro-organisms in relation to some Diseases of Women.'

[3] Ballantyne, 'Malformations of the Genital Organs,' Allbutt and Playfair, *op. cit.* p. 63, with complete lists of recent references.

[4] Ballantyne, 'Hermaphroditism,' *op. cit.* p. 100, and references, p. 111.

CHAPTER VI

CATARRH OF THE CERVIX, EROSION, ENDOMETRITIS

Catarrh of the cervix, perhaps one of the commonest diseases of women, is better described as cervical endometritis: that is, an inflammation of the mucosa associated with chronic inflammation of the cervix itself. Endocervicitis and cervicitis are other terms employed, but they are unnecessarily complicated.

Catarrh of the cervix is a generally chronic disease; of course, acute catarrh may occur, especially with cases of septic infection associated with injury, such as labour or abortion, or the use of septic tents or dirty instruments, and uterine 'tinkering' generally. It may arise as an extension of an acute infection from below, simple possibly in a few cases, but generally puerperal or gonorrhœal. This soon passes on to a general endometritis, *i.e.* endometritis of the body of the uterus, and if acute enough, as in puerperal cases, it extends to the tubes and peritoneum.

It is difficult to talk of inflammation of the cervix apart from the body of the uterus; yet in some cases, especially in gonorrhœa, the brunt of the inflammation falls upon the cervical tissues. In such cases the cervix is swollen and there is acute catarrh with abundant purulent secretion, but even in these it is difficult to eliminate the possibility of the discharges coming from the body of the uterus itself.

Chronic catarrh of the cervix is a very different matter. It presents the usual appearances of the catarrhal condition; the mucous membrane is swollen, red, and easily bleeds, and secretes a muco-purulent fluid; often the secretion is nearly healthy, yet the widest variations take place, and the point seems to be, what secretion shall be called morbid and what shall be called natural? The normal secretion of the glands of the cervix is tenacious, viscid, clear, alkaline mucus. A milky white discharge in small quantity is not morbid, but a yellow or purulent discharge surely indicates disease. Probably the normal secretions in great excess may be termed pathological. Then again, great confusion exists as to what is meant by the word ' discharge. ' Leucorrhœa[1] or ' the whites ' commonly means any discharge from the vulva which is not blood, and since the body of the uterus, the cervix, the vagina and the vulva all have their normal secretions, a knowledge of these and of their exact source and normal characters is necessary, before we can term a discharge pathological. Nor have we any exact knowledge of the quantities of these secretions in health, or their variations with age. Patients do not notice a discharge until it becomes extraordinary or excessive, and yet with extensive disease there may not be excessive discharge, and the reverse may hold good. All discharges should, if possible, be traced to their source ; this is often a difficult matter, but in chronic inflammation of the cervix a speculum should show the diseased surface, or the discharge coming from the canal. The vaginal portion is often swollen and the canal is widened ; there is frequently erosion, of which more will be said presently, and the secretion is often muco-purulent. There is much glandular hypertrophy and overgrowth, and the tissues are infiltrated with leucocytes. Practically, changes take place very similar to those which are included in Endometritis proper, where they will be found described.

Lacerations of the cervix are frequently found accompanying inflammation and catarrh of the cervix, and are as a rule produced in labour, either ordinary or instrumental, frequently the latter. They are generally lateral, they may be multiple ; and may extend upwards to the os internum, or down the vagina, or deeply into the cellular tissue.

Care must be taken in recognising the difference between the condition called ' erosion,' and exposure of the red cervical mucous membrane due to a laceration (' ectropion '). When such lacerated portions of the cervix are drawn together the ectropion disappears, whereas the patch of redness called erosion would not so be altered.

Much has been written about the results, immediate and remote, of tears of the cervix. They are probably unimportant and have few symptoms, certainly no reflex ones, nor does there appear to be any relation between tears of the cervix and cancer. If there be severe bleeding from a torn cervix in labour, or after some operation such as dilating, or where the cervix has been divided intentionally for the removal of some intra-uterine growth, such a tear may be sewn up: but the planned operation of sewing up every torn cervix that can be found is rarely needed. One of the commonest conditions found with cervical catarrh, and probably depending on it, is **erosion,** [1] a name given to the red granular surface so commonly seen around the os externum. Erosion used to be called an ulcer, [2] *i.e.* a loss of tissue, but on the contrary in erosion there is an increase of tissue, and the condition should be described as a sessile adenoma. The vaginal portion of the cervix is normally covered with many layers of squamous epithelium continuous with that of the vagina, and at, or about, the os externum, the epithelium becomes suddenly cubical and is in fact the columnar glandular epithelium which lines the cervix and body of the uterus, consisting of a single layer of tall cells. These cells are continuous with the glands of the cervix, which are very numerous, and which are also lined with a single layer of columnar epithelium. Towards the canal of the cervix itself the epithelium is ciliated. The glands of the cervix are more complicated and larger than the glands of the body of the uterus; in fact, the cervix is itself a gland, and is a structure quite apart from the body of the uterus.

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[1] Herman, *Diseases of Women*, p. 424.

It is in connection with these glands, or at all events with the columnar glandular epithelium of the cervical canal, that the interest lies in the pathology of erosion. It is now undoubted that in this condition the red patch is due to an overgrowth and hypertrophy of the gland tissue of the cervix replacing, or actually growing through, the squamous epithelium which normally covers the vaginal portion. Normally, by the speculum, the layers of squamous cells covering the vaginal portion appear opaque and pink, the blood-vessels beneath are covered by many layers of cells more or less opaque, and exceedingly tough; but when the surface of an erosion is seen it appears bright red, as it is only covered by one layer of columnar cells, the bright colour of the underlying blood-vessels showing through. Whether these columnar cells are derived from proliferation and extension downwards of the glands themselves through the squamous epithelium, or whether the columnar epithelium creeps down and round the margins of the external os, and so displaces the cells covering the vaginal portion, is not a matter of great importance ; the fact is still the same. Erosion is not an ulcer, but an increase of tissue ; in fact, an adenoma of the cervix.[1] Careful sections of these bright red patches on the cervix show their true nature ; they consist of masses of gland acini, either cut longitudinally or in cross section, such gland tissue being identical with the mucous glands of the cervix itself. The stroma is generally simple, with some inflammatory exudation ; in some cases the section exhibits a great quantity of glandular elements, or the simplicity of the glands is exaggerated and they are increased at the expense of the stroma, with wide convolutions and dilatations. In other cases these dilated glandular acini form cysts, and masses of such mucous cysts may occupy the surface of an erosion or extend deeply into the tissues of the cervix itself. These small tense cysts are quite commonly seen on the surface of an erosion, and if pricked with a knife a drop of clear tenacious mucus escapes. These are termed Ovula Nabothi, and the condition may be spoken of as cystic, or **follicular erosion**. Sometimes a large mucous cyst may occupy the tissues of the cervix, giving rise, before it is punctured, to the suspicion of a solid growth, or even malignancy. At other times the overgrowth of glandular elements seems to be small, while that of the interstitial tissues of the stroma is great, and we get an enlargement of the cervix by a growth containing cysts exactly resembling ordinary mucous glands of the cervix, really a cystic adenoma.

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[1] The varieties of Erosion may be termed:—1. Simple; 2. Papillary; 3. Follicular.

[2] Herman, *Diseases of Women*, p. 446.

Apart from these tumours of the cervix, which will be spoken of later, the surface of an erosion may also develop very rapidly towards the free surface ; it then soon becomes papillary in its nature, and raised above the surrounding surface. In these cases the glandular hypertrophy is very great, accompanied with increase of blood-vessels, forming the so-called **papillary erosion.**

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[1] Ruge and Veit, *Zeits. f. Geb. u. Gyn.* 1878, vol. ii. p. 415 : 'Zur Pathologie der Vaginal-Portion.'

The stage of adenomatous overgrowth may go further, and the mass becomes stalked, with an axis of fibrous tissue and blood-vessels, and we have the formation of a **mucous polypus.** This may be a quite simple pedunculated adenoma, consisting of glands exactly like those of the cervix, with a stroma greatly myxomatous, and with large vessels, some mucous polypi being very vascular. The polypus may take on the cystic type,[1] or the whole may be racemose, and hang in a grape-like cluster from the cervix; such are rare. Some of the grape-like polypi of the cervix are not of this character at all, but the worst forms of sarcoma. Rarely, these adenomatous polypi of the cervix have wide dilated channels in them, which are either ruptured cystic cavities or the remains of dilated glands: they are called *channelled polypi.* Œdema is common. Mucous polypi, projecting into the vagina for any length of time may be covered with squamous epithelium, which appears to replace the columnar which originally covered them. A similar change takes place on the surface of a fibrous polyp hanging through the cervix.

Some so-called polypi are merely projecting masses of carcinoma; in fact, it is well to examine carefully such growths when removed, as they are not always of a simple character.

Endometritis proper.—Nothing, perhaps, is more difficult to define than ‘Endometritis’ [2]—so much is included under the term. Some authors limit the meaning to mere inflammatory conditions, others to overgrowths of the endometrium, others to atrophy of the endometrium, others to inflammations not only of the endometrium but of the whole uterus. Nothing is more puzzling; even the normal conditions of the endometrium are more or less unknown; the endometrium is difficult to see, and we know little of its changes either between or during the menstrual periods (see Dysmenorrhœa); still less do we know of the changes we term pathological.[3] We speak of discharges from the uterus being normal and pathological, and therefore indicative of health or disease, but the subject is of deep obscurity; mere increase or ‘whites’ may not be disease, yet profuse, foul, purulent discharges certainly represent pathological conditions.

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[1] Herman, *Diseases of Women*, p. 456.

[2] Herman, *Clin. Journ.* April 15, 1896.

[3] Champneys, *Painful Menstruation*, London, 1891.

What are the conditions included under the term Endometritis in text-books?[1] Herman[2] describes very well what is known of the subject, and his account will be followed in the present work. Endometritis, in the ordinary sense of the term, means inflammation of the endometrium[3]; it is more or less transitory, but its importance lies in the fact that it may extend to the Fallopian tubes and peritoneum. Such may be the result of any infection, small or great; its importance certainly becomes great when we speak of puerperal infection, small when it gives rise to no symptoms beyond a mucopurulent discharge, and when the lining membrane of the uterus shows only slight evidence of inflammation.

Still this does not include all the conditions known as Endometritis: a large class included in text-books are those associated with great hypertrophy or overgrowth of the endometrium, the so-called 'Villous or Fungous endometritis.' Such conditions are really not due to an 'itis' at all; they probably are not inflammatory, but should be classed under adenoma of the body of the uterus; some even touch upon malignancy.

Again, another group of diseases called endometritis are associated with atrophy, the 'atrophic endometritis' of some writers. So we have under the heading **Endometritis**:[4]

a. True inflammation of the endometrium.
b. Conditions associated with overgrowth of endometrium.
c. Conditions associated with its atrophy.

True Inflammation of the Endometrium.—This is Endometritis proper, and its greatest importance lies in the fact that it is one of the phenomena in the spread of septic infection upwards to the tubes and peritoneum, and as such is only a stage of general infection which in the end leads to the fixation and tenderness of the uterus, pelvic peritonitis and cellulitis, general peritonitis and death in severe puerperal infection; and to pyosalpinx and sterility, in less marked cases. Great confusion exists also on the question of the use of the term 'Metritis,' at all events in acute infection; but here the word metritis will not be used apart from acute endometritis. No doubt acute general inflammation of the uterus does occur in puerperal infection, with abscesses and sloughing of the cellular tissue, and the uterus is enlarged, painful and tender. Probably under the term 'chronic metritis' is also included Subinvolution.

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[1] Ruge, 'Endometritis,' *Zeits. f. Geb. und Gyn.* vol. v. p. 317.

[2] Herman, *Diseases of Women*, p. 458.

[3] Pozzi. *Endometritis and Metritis*, New Sydenham Society, translation, 1892, vol. i.

[4] Herman, *op. cit.*

Generally, in **acute endometritis** the whole mucosa is inflamed; there is great increase in the glandular elements, with infiltration of leucocytes into the connective tissue; there is great cell proliferation, and the glands are swollen, especially near the surface. Degeneration of epithelium then takes place, and the tissues are found crowded everywhere by small round inflammatory cells, accompanied with hæmorrhage, while some of the vessels themselves appear to undergo degeneration. In the worst cases the inflammatory changes are so acute that the whole endometrium is destroyed or sloughs, the deeper tissues of the uterine wall are involved, and the muscular tissues infiltrated everywhere with inflammatory cells, which in places break down into abscess cavities. Veins and lymphatics are involved, and the disease spreads to surrounding peritoneum or cellular tissue. The whole uterus is enlarged, tender, and possibly fixed, the tissues of the broad ligaments become involved, and septic infection travels to pelvic veins and lymphatics, with a more or less rapidly fatal event. Other cases are less severe; the disease becomes chronic, with constant discharge, or, if the cause be removed, the patient may recover completely.

Causes of Endometritis.[1] (*a*) *Puerperal Infection.*—This may be very severe, as in Puerperal Fever cases, and the infection so rapid that death ensues within a few days, or the disease may run a more chronic course.

Frequently it arises with some retained and decomposing products of conception after labour and abortion. The importance of throughly emptying the uterus in such cases, and of taking precautions against infection, cannot be too strongly urged. Much depends upon the severity of infection, whether local or general; removal of the cause in sapræmic cases results in recovery, but systemic infection is a very serious matter. The subject belongs rather to obstetrics than gynæcology, but septic endometritis is often the origin of a more serious general infection. The condition is to be suspected after labour when the lochia are putrid, or pus pours from the uterus, or if the uterus is too big, with marked tenderness, and where the involution curve remains high.[1] It must not be forgotten that the discharges in the vagina may be foul and the uterus itself sweet, or at other times the vagina is not foul, yet, on introducing the finger into the uterus, its cavity is putrid ; and again, in some of the severest septic cases the discharges are not necessarily foul at all.

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[1] Herman, *op. cit.*

Perhaps the greatest importance of these cases of endometritis is their relation to subsequent diseases of the tubes and peritoneum.

Endometritis may also run a more or less *chronic* course, with constant discharges ; such cases are seen in the cervix associated with laceration, eversion, and erosion, and in the body of the uterus, in connection with tumours, such as necrotic fibroids (fig. 62) and polypi, and with cancer. Here the source of the endometritis is the breaking down of these products, which become infected and gangrenous, much the same way as do the retained products of conception.

Septic infection of the endometrium also occurs as the result of operations with unclean instruments, tents, dilators, stem pessaries and sounds. The too frequently used curette is often responsible.

(*b*) *Gonorrhœa*[2] is responsible for some cases of endometritis, mostly cervical, and the infection once lodged in the cervix is difficult to eradicate ; there is frequently erosion and purulent discharge. The importance of gonorrhœa is not so much its relation to mere endometritis, as that the endometritis is but a stage of infection of the tubes and peritoneum, with resulting salpingitis, pyosalpinx, and sterility, from sealing up of the tubes.[3]

(*c*) *Endometritis associated with Febrile Disease.*—This may occur in cases of typhoid fever, cholera, measles, smallpox, and scarlet fever ; probably the endometrium shares in the febrile changes, and anomalies of menstruation occur, sometimes with discharge and hæmorrhage. Such an endometritis is quite subordinate to the disease which produces it.

Tubercle of the endometrium occurs, and as such has some relation to endometritis, but whether it is to be regarded as a primary infection or part of the general constitutional condition is at present uncertain. Very little is known of the early stage of tubercle of the endometrinm. Most museum specimens[1] exhibit general infiltration of the whole uterus, tubes, and surrounding peritoneum, with caseous cavities which are breaking down, and we are unable to say whether the cases began in the endometrium or not; isolated nodules of tubercle can be seen affecting both endometrium and the uterine wall, and tubercle with definite giant cells and epitheloid formations are seen in scrapings. Later in the disease the glands more or less disappear or are choked with inflammatory cells; in advanced cases the whole of the interior of the uterus is covered by necrotic tubercular matter, the process of ulceration spreading to the muscular layers. Bacilli[2] are hard to find. Tubercular endometritis is seen sometimes in children and young adults; it is probably a part of general tuberculosis, but in some cases the tubes and peritoneum appear to be affected later than the endometrium.

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[1] Griffith and Lea, *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 246.

[2] See chapter on 'Gonorrhœa in relation to Pelvic Inflammation.'

[3] Cullingworth, *Brit. Med. Journ.* July 20, 1889.

The relation of micro-organisms to endometritis is of great interest, and the part played by streptococci in puerperal sepsis[3] is of vast importance, less so that of the gonococcus and tubercle bacillus. In health no micro-organisms should exist in the uterus,[4] and even in disease their presence may be accidental. On the other hand, many foreign observers attach great importance to the micro-organisms as a cause of endometritis.[5] Winckel classifies endometritis into two definite groups[6] as follows:

(*a*) *Endometritis due to micro-organisms.*
  (1.) Gonorrhœal.
  (2.) Tubercular.
  (3.) Puerperal.
  (4.) Saprophytic.
  (5.) Diphtheritic.

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[1] Kelly, *Operative Gynæcology*, vol. i. p. 489, and P. D. Turner. 'Notes on Tuberculosis of the Fallopian Tubes and Uterus in cases of Phthisis,' *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 359; see also p. 344.

[2] Turner, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 161, plate 4.

[3] Pfannenstiel, 'Zur Aetiologie des Puerperal Fiebers,' *Centralb. f. Gyn.* 1888, p. 617.

[4] Döderlein, 'Micro-organisms in the Lochia,' *Centralb. f. Gyn.* 1888, No. 23.

[5] Winckel, *Centralb. f. Gyn.* 1895, No. 26.

[6] Freeland Barbour, Allbutt and Playfair's *System of Gynæcology*, p. 206.

(6.) Syphilitic.
(7.) Due to fungi and amœbæ.

(b) *Endometritis not due to micro-organisms.*
(1.) Simple catarrh.
(2.) Hæmorrhagic.
(3.) Decidual.
(4.) Exfoliative.

Chronic Endometritis frequently accompanies subinvolution; both are often due to a common cause, and probably most cases formerly described as chronic metritis[1] come under the heading of subinvolution. In **Subinvolution,**[2] the uterus is enlarged throughout, the cervix is thickened, the cavity is larger and broader, but its anterior and posterior walls are in contact. A subinvoluted uterus is not necessarily a diseased uterus, though inflammatory conditions of the endometrium sometimes exist, but this is not always the case. The condition has been ascribed to a general diffuse overgrowth of connective tissue which replaces more or less the muscular elements of the uterus.

Subinvolution is due to want of perfect contraction of the uterus,[3] by which normal involution is retarded; therefore, whatever hinders involution produces subinvolution. Want of proper uterine contraction is seen when the uterus is not emptied after labour, where it is full of clots, or when membranes or placenta are retained. It occurs after post-partum hæmorrhage, with septic infection after labour, and especially with pelvic inflammation. Multiparity is also a factor in subinvolution.

Normal **Involution** is a physiological retrogression.[4] There is no pathological atrophy and degeneration of muscular fibre with increase of connective tissue. The process is chiefly one of simple shrinking and diminution of the muscular fibres. There is no fatty change, nor increase in connective tissue corpuscles. Helme thinks the process rather a chemical one, a sort of solution (peptonisation ?),[4] the contents of the muscle cells becoming more soluble. They become cloudy, instead of translucent as in pregnancy ; they rapidly diminish in volume ; then a more gradual shrinking follows, the large fibres becoming small. There is no karyokinesis. As regards the connective tissue, it becomes granular and then disappears. Large leucocytes or giant cells are seen in the first few days of involution. In the subperitoneal connective tissue some fat cells are produced. Vascular changes consist in compression and obliteration of vessels, as they are no longer wanted. Eden[1] observed thrombosed vessels in the uterine wall long before the placenta was ripe. Larger vessels are affected with true endothelial proliferative arteritis and conversion into fibrous tissue, and an increase of fibrous tissue takes place in the walls of the vessels and around them, so that at the end of involution many such vessels can be plainly seen in a section of the uterus as opaque projecting yellowish spots. Sir John Williams[2] believes this to be absolutely indicative that pregnancy has taken place.

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[1] The author does not propose to describe Metritis apart from Endometritis.

[2] Herman, *Diseases of Women*, p. 88, ‘Morbid Anatomy of Subinvolution.’

[3] Sir J. Williams, ‘Subinvolution of the Uterus,’ *Brit. Med. Journ.* vol. ii. 1882, p. 405; and Ries, *Zeits. f. Geb. und Gyn.* vol. xxiv., quoted by Herman, *op. cit.* p. 89.

[4] T. A. Helme, *Trans. Royal Soc. Edin.* vol. xxxv. part ii. No. 8.

Superinvolution.—This means that involution goes beyond the stage of restoration of the uterus to its proper size ; it leads to permanent diminution of the size of the organ, and arrest of its functional activity.[3] It should be called Puerperal Atrophy ;—in such a case the uterus is all too small, its walls and the muscular tissue are thinned, its mucous membrane is scanty or absent, while in the walls thrombosed and obliterated vessels are seen. The condition may follow on any puerperal illness, such as suppuration and destruction of the ovaries, or pelvic cellulitis (which contracts the vessels). It may occur with severe puerperal cachexia, and wasting or inflammation of the uterus severe enough to destroy the inner part of the wall (endometritis dissecans).[4] Ovarian atrophy appears to be a constant factor, with permanent amenorrhœa, and sterility, and there is an early and permanent menopause. It is incurable.

(2.) **Endometritis associated with Overgrowth.** (Figs. 17, 18.)—This condition is not endometritis, as such, at all ; inflammation probably is not a factor in its causation ; it is a condition of hyperplasia, hypertrophy and overgrowth of the endometrium,

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[1] 'The Ripe Placenta,' *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 360.

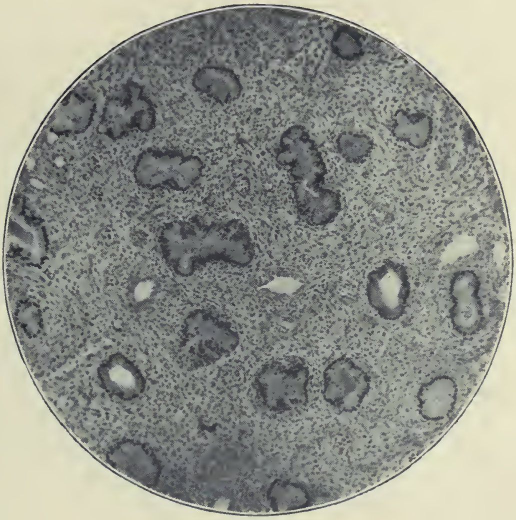
[2] Sir J. Williams, *Trans. Obstet. Soc. Lond.* vol. xx. 1878, p. 172.

[3] Herman, Allbutt and Playfair, p. 447.

[4] Herman, *ibid.* p. 448.

Fig. 17.

SIMPLE ENDOMETRITIS.

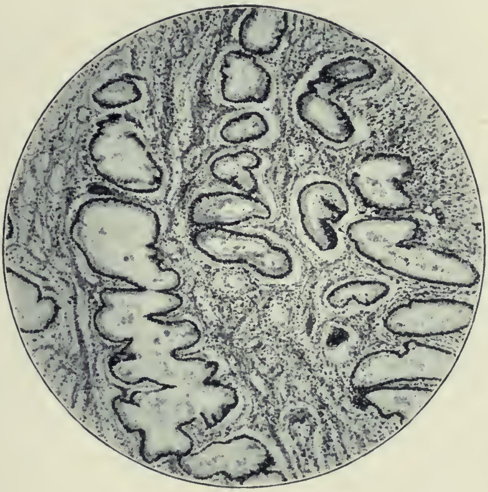


Section of portion of endometrium removed by the curette in a case of so-called 'endometritis.' The uterus was enlarged. There had been much irregular hæmorrhage. The endometrium was thickened and vascular. The section shows marked increase of the glandular elements of the mucosa in transverse section, but they retain the type of simple uterine glands. The stroma is more vascular than normal, and there is small-celled inflammatory exudation. The condition is really one of adenoma of the body of the uterus.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 18.

SIMPLE ENDOMETRITIS.

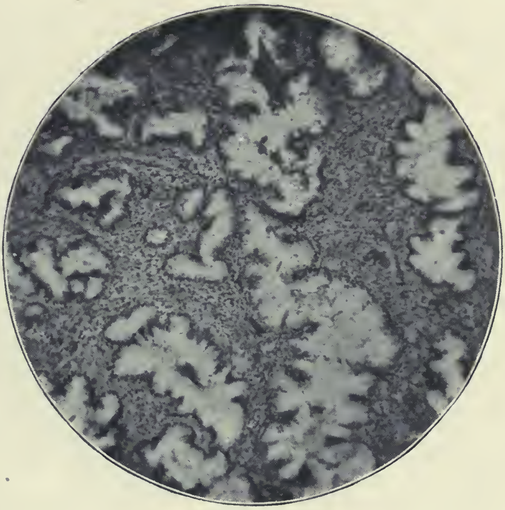


Longitudinal section of portion of endometrium removed by the curette in a case of so-called 'endometritis.' The endometrium is much thickened and the glandular elements increased both in number and complexity, but the likeness to ordinary uterine glands still remains. The stroma is vascular, and has undergone myxomatous changes. The condition is really one of adenoma of the body of the uterus. Sections of mucous polypi of the body of the uterus show changes identical with the above.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 19.

SIMPLE ENDOMETRITIS.

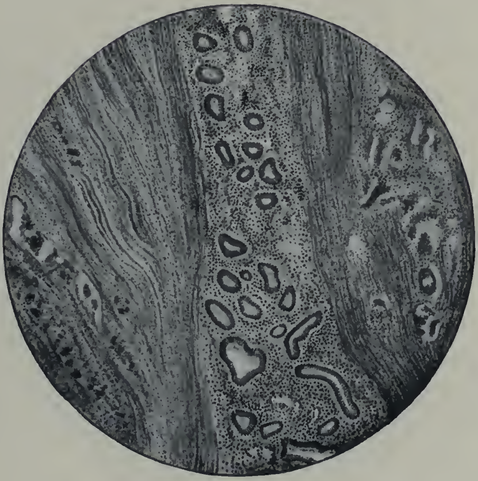


Section of endometrium of a uterus, showing the more advanced stages of simple endometritis (adenoma of the body of the uterus). The mucosa is very thick, and there is great glandular proliferation of a simple nature. The section shows convoluted gland spaces of great complexity, lined with a single layer of columnar epithelium. The epithelium is regular, and does not involve the surrounding stroma. (High power.)

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 20.

ADENOMA OF THE BODY OF THE UTERUS.

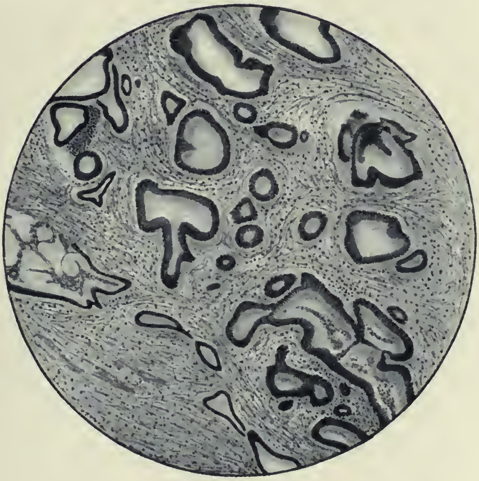


Section through uterine tissue at the site of growth, from the same case as in fig. 21, showing penetration of gland tubules between the fibro-muscular bundles. In its general characters the case seemed non-malignant, but the epithelial proliferation and the penetration of tubules into the uterine wall are points in favour of malignancy. The patient, a nullipara of 49, died suddenly of pulmonary embolism on the fourteenth day after operation, so no subsequent history is possible.

(Eden, 'Trans. Obstet. Soc. Lond.' vol. xlii. 1900, plate i. p. 2.)

Fig. 21.

ADENOMA OF THE BODY OF THE UTERUS.



Section of tissue removed by the curette, showing regular gland tubules and abundant fibro-muscular stroma. Sections of the uterine wall are shown in fig. 20.

(Eden, 'Trans. Obstet. Soc. Lond.' vol. xlii. 1900, plate i. p. 2.)

a new formation, and the name 'Adenomatous Disease of the Body of the Uterus' is a more correct term. It is a new growth, and in some cases not of a simple character; in fact, some cases described as adenoma pass insensibly over the border line and become malignant.[1]

Existing accounts and names for this disease are very confusing. Two distinct types appear to exist:

(*a*) **Polypoid.**—The endometritis polyposa, fungous or villous endometritis, or metritis interna villosa of other authors.

In this condition the endometrium becomes hypertrophied and thickened, and consists of masses of adenomatous tissue made up of glands which in a way imitate the normal uterine glands, but themselves run riot in their overgrowth, yet do not necessarily pass the stage of innocency. Great proliferation, tortuosity, and even cystic dilatation of the glandular structures are often found (fig. 19), with general increase of connective tissue, new vessel formation with liability to hæmorrhage, and marked myxomatous changes. In some specimens the glandular elements are most in evidence; in others, the connective tissue. Sometimes only local areas of the mucosa are affected, and the growth is localised and projects, as a soft, very vascular mass, into the cavity of the uterus, becoming pedunculated. Such growths are really mucous polypi of the body of the uterus; in other cases, large areas of the mucosa take on this polypoid or villous type of adenoma. Sections of such pieces of growth removed by the curette are most puzzling and often misleading. A mere piece from the surface of such a growth may appear of a simple glandular type (an adenoma benignum), but the implication of the walls of the uterus by glandular invasion may be going on, and although the glandular elements may still be retained within the acini, such groups of gland elements invading muscular tissue are to be looked on with extreme suspicion. It is difficult to get pieces of tissue by mere curettings to follow out such investigation, but clinical evidence is sometimes forthcoming in that the disease returns again and again, and it is more than probable that such growths have ceased to be benign and are essentially malignant adenomata, coming under the head of cancer of the body of the uterus.[2] (Figs, 20, 21.)

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[1] See chapter on 'Cancer of the Uterus.'

[2] See *ibid.*

Enlargement of the body of the uterus in such cases in sterile nulliparous women should suggest cancer rather than adenoma. Other cases, where connective tissue elements are chiefly in evidence and the disease recurs, are sarcomatous.

(*b*) **Diffuse or Hyperplastic.**[1]—In this there are no localised outgrowths from the endometrium, the whole mucosa is uniformly thickened and is extremely vascular, and there is often profuse pink watery discharge, which is not offensive. Sections of the endometrium resemble that of the Polypoid variety ; there is marked glandular overgrowth, with great thickening of the whole lining membrane of the uterus, sometimes to the extent of a quarter of an inch or more. The glands are caricatures of the normal uterine glands and are very complex. Hæmorrhage into the endometrium is common, and there is great increase of new vessels. Little is known of the condition ; it is not inflammatory. Examples of this are seen best in uteri containing fibroids[2] or fibroid polypi ; the name 'endometritis' is often given to the thick mucosa found with uterine fibroids, but it is probably incorrect ; inflammation of the endometrium may occur, but only if such a fibroid is dead or sloughing.

More will be said of this in the chapter on Uterine Fibroids, but the watery discharge and the severe hæmorrhage that occur in such cases come from this enormously hypertrophied endometrium. Section of a fibroid uterus occupied by submucous fibroid tumours shows that over the surface of the tumour,[3] and in the corresponding surface of the opposite wall where such a tumour impinges, the endometrium is thinned and atrophied from pressure, while at the base and surrounding the protruding mass the endometrium gets thicker and thicker till it blends with that of the rest of the cavity of the uterus. At the edges of this 'collar,' so to speak, of endometrium, the blood-vessels and capillaries are very numerous, and many of them are ruptured. Exactly the same condition exists in the pit on the opposite wall into which the fibroid fits.

Some of the bleeding in submucous fibroids comes from these recently ruptured vessels in this 'collar' of tissue from which the fibroid gradually protrudes.

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[1] Olshausen, 'Ueber chronische hyperplasirende Endometritis des Corpus Uteri,' *Archiv f. Gynäk.* vol. viii. pt. i. ; Heinricius, *Arch. f. Gyn.* vol. xxviii. p. 163 ; Freeland Barbour, Allbutt and Playfair, *Inflam. of Uterus* ; Herman, *Clin. Journ.* April 15, 1896.

[2] Wyder, 'Die Mucosa Uteri bei Myomen,' *Arch. f. Gyn.* vol. xxix. p. 1.

[3] H. Kelly, *Operative Gynæcology*, vol. i. p. 539.

In fibroid uteri, large mucous polypi of the body (pedunculated adenoma) are not uncommon. Removal by the curette of such thickened endometrium, or of these polypoid masses, may check more or less the hæmorrhage in such cases. This condition of endometrium is sometimes seen in heavy subinvoluted retroverted uteri. Sterility seems to accompany this endometritis, but whether as cause or effect is uncertain.

(*c*) **Endometritis with Atrophy.**[1]—This is sometimes called 'Senile Endometritis,' as it commonly occurs in old women: here the condition is the reverse of the hyperplastic form, and the curette only removes small fragments of tissue which exhibit gland structures in a process of degeneration, or no glands at all may be found. Patches of squamous epithelium may occur; very few vessels are found, and they are degenerate and thickened, or obliterated.[2] The connective tissue is generally in excess of gland tissue, and the whole mucosa may be replaced by fibrous tissue; the uterus is not enlarged. There is often a pink watery discharge; it is sometimes offensive[3] or even purulent. The condition appears to be one of senile degeneration, and the decomposition of the contents of the uterus only accidental; others are more suspicious and run the course of cancer of the body.

Sarcoma and carcinoma of the endometrium are treated of under Cancer of the Body of the Uterus.

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[1] Herman, *Diseases of Women*, p. 322.

[2] Cornil, *Anatomie Pathologique des Métrites.*

[3] Winckel attributes the decomposition to cocci and bacilli.

CHAPTER VII

UTERINE DISPLACEMENTS

These may be divided into two classes :—

a. **Minor Displacements.**
  1. Anteflexion and anteversion.
  2. Retroflexion and retroversion.
  3. Lateri-version.

b. **Major Displacements.**
  1. Descent.
  2. Retroversion of the gravid uterus.
  3. Inversion.
  4. Pendulous belly of pregnancy.

Minor Displacements.[1]—The importance of minor displacements of the uterus is very small, and in fact, though so much has been written on the subject, it must be remembered that minor displacements are not diseases at all, nor do they necessarily give rise to any symptoms whatever, either immediate or remote ; because a uterus is a little bent one way or the other, it is not necessarily diseased. Again : What is the normal position of a healthy uterus ? Anteflexion is the natural position of every uterus in all women ; child-bearing may make the shape straighter, but that is all. A full bladder may interfere with this natural position of anteversion, but when it is empty the uterus will be in a position of anteversion.

Much has been made of **Pathological Anteversion** ; it is said to occur with parametritis of the utero-sacral ligaments ; it is unimportant. Again, anteversion is said to be congenital. Some ill-developed uteri are very much anteverted or flexed ; here it is not the flexion that is important, but the ill-development ; this was referred to in the chapter on Dysmenorrhœa.

Lateri-version is unimportant ; a uterus a little to one side or the other is not a disease. If such lateral, forward, or backward displacement be caused by some tumour or adhesion, it is the tumour or adhesion that is important, not merely the displaced womb.

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[1] Matthews Duncan, *Clin. Lect.* 4th ed. pp. 406, 412.

Retroflexion,[1] *i.e.* the cervix in the natural position and the body bent back, and **Retroversion,** *i.e.* the body back, the cervix forwards, the uterus itself being straight, are perhaps of some importance, but even then the only importance is that they are stages of descent of the womb, which is quite a different matter. Another kind of case exists frequently where the uterus is found retroverted and somewhat enlarged, and where the appendages are prolapsed and tender, the latter causing at times great suffering or interference with bladder and rectum. Here it is not so much the mere position of the uterus which is important as the descent of the appendages, which are sometimes fixed by pelvic peritonitis in Douglas's pouch ; in these cases the uterus is bulky, heavy, and bleeds easily. The condition often depends upon want of care of a patient after parturition : the uterus is allowed to remain more or less retroverted by injurious pressure from above, and subinvolution is common, whereas after delivery anteversion is, and should be, the proper position of the womb. Very often, after delivery, such retroverted uteri have not been properly emptied, and are a source of trouble and hæmorrhage afterwards. Sudden retroversion in virgins may occur ; it is very rare.

Looking at another matter, it is often stated that flexions cause obstruction, *i.e.* the uterus is so bent that the canal is kinked, and the contents of the uterus cannot get out. This is untrue ; it is 'fireside pathology' : no specimen in any museum shows such a thing, or at least the author of this book cannot find it, though it is seen in diagrams and pictures. The uterus is a thick-walled tube and will not kink or form a spur in the canal, or ever obstruct the menstrual flow. This was referred to in the chapter on Dysmenorrhœa.[2]

Neither do flexions and versions cause congestion of the uterus. The uterus is supplied with circular blood-vessels,[3] which are offshoots of the main lateral trunks ; these encircle

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[1] Generally, Retroversion and Retroflexion are combined.

[2] See also Champneys, *Painful Menstruation*, Lond. 1891, p. 60 ; Harveian Lectures, 1890.

[3] Sir J. Williams, 'On the Circulation in the Uterus, with some of its Anatomical and Pathological Bearings,' *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 112.

and supply each segment of every part, so that even the tightest constriction would not impede the blood supply above or below it: how much less would a mere flexion do so!

Pressure symptoms are not, and never will be, produced by mere flexions. The weight of the normal uterus is small enough: it can be sent by post for a penny; yet within the healthy abdomen its weight is less still. The uterus is a floating organ like a cork; the abdominal pressure is negative; everywhere the womb is supported. What pressure symptoms can it cause if it be healthy? It causes none.

Patients only know of these 'Minor Displacements' when they are told. Once tell a patient about her 'inside' and that she has a 'displacement'; she never forgets, though she has no symptoms, the displacement remains in her mind: hence it is her mind that needs treatment, not the displacement. Even major displacements may cause no symptoms at all, and yet the minor are said to cause every symptom under the sun.

The association of neurasthenia with minor displacements [1] is seen, but it is not cause and effect; both are common causes produced by functional weakness. Minor displacements are akin to flat foot and lateral curvature.

Sterility [2] is not the result of displacements. Anteflexion and retroflexion exist with sterility it is true, especially anteflexion, but in such cases the real cause of the sterility is imperfect development of the uterus, which frequently co-exists with anteversion.

The whole matter of minor displacements is well summed up by the late Dr. Matthews Duncan as follows ('Clinical Lectures,' 4th ed. p. 407–411):—

'No subject seems to have occupied the attention of gynæcologists more than this. Flexions are perhaps themselves important conditions of other diseases, but not in themselves disease. A verted or flected, ante, retro, or laterally placed womb is not a diseased womb, nor does it cause any pain, or disorder, or disability. You will not be able to comprehend the difficulties of this matter, nor to appreciate it justly, unless you include in your judgment the influence of enthusiasm on the doctor, and enthusiasm and hysteria on the patient; both are so delighted with the simplicity and intelligibility of the supposed disease, and so impressed by them, that they are with difficulty made content while the displacement continues, even although pains and aches and every known evil are gone. Treatment has no rational end, for in spite of all treatment the displacement does continue, and, were such doctrines that all evils possible are due to displacements well founded, life for women would not be worth having, for the position of no womb satisfies those who entertain them, and treatment has as its ordinary consequence failure and disappointment, and sometimes grave disaster.

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[1] Herman, *Diseases of Women*, p. 108.

[2] Herman, *op. cit.*, p. 638; *id.* 'On the Relation between Backward Displacement of the Uterus and Sterility and Abortion,' *Trans. Obstet. Soc. Lond.* vol. xxxiii. 1891, p. 499.

'Flexion and version may be useful terms, but all displacements are forms of descent: that is, not only a bit of the womb or the womb alone that descends, but the whole of the abdominal contents as well, or at least the contents and part of the soft tissues of the pelvis. The descent, so far as it affects the womb, may move one part more than another, and then you have a flexion; or it may move the whole, and then you have a version or a descent.

'Flexions, versions, and descent are not necessarily the cause of any discomfort or disorder. Thousands of women have displacements; to treat a displacement merely because it exists is a gross error, and yet not a rare one. Such a simple uncomplicated displacement is not a disease; it is the condition of equilibrium of that woman's pelvic viscera; and therefore that displacement is a constituent part of her comfort and health.

'A woman may have her uterus not only displaced, but monstrously mis-shapen or distorted by a fibroid or fibroids, and yet not have a single pain or ache or any discoverable disorder. In fact, it would be hard to say what shape and what position of the womb are unnatural, not to say morbid; certainly its shape and position have a very wide range within the limits of the natural or not abnormal.

'There is a vast number of cases of chronic disordered health in women, of most varied kinds, which are associated with displacement and descent, and it is very common in the present day to regard the displacement as important or as the chief factor in these cases. Even when there are no local or direct symptoms whatever, the displacement in such cases is trivial not demanding treatment.

'You may have the displacement without symptoms, and the symptoms without displacement, and till we have some evidence better than we have now, that the symptoms depend on the displacement, I advise you to leave the displacement alone; you cannot successfully treat it, and if you did, your patient is not nearer to restored health.

'The symptoms of displacement and descent cannot be definitely described, nor is there any relation between the degree of displacement and the severity of the symptoms; they are never the result of the displacement, pure and simple.

'There is no satisfaction or rationality in the common explanation founded on pressure, forwards in one case, or backwards in the other.

'If there is no complication by adhesion or otherwise, pressure forwards and pressure backwards are, in every case, just the same—equal and contrary. Complications, then, are generally the chief matter in displacements, not the displacements *per se*, and though the complications are generally congestive, and inflammation of the womb or of the parts of it, or of the neighbouring organs. To guide you in cases of this kind, you need wisdom over and above, but not without, science and experience.'

ON DESCENT OF THE WOMB

Under descent of the womb are included several things. The womb may come down partially, **Prolapse**, or the whole organ may fall forth from the body, **Procidentia**; this is a mere matter of degree and is more or less unimportant. Or again a portion only of the womb may protrude at the vulval orifice, and be in a condition of descent, while the body of the uterus itself is in a more or less normal position; in such cases an elongation of some portion of the cervix has taken place.

Congenital elongation of the vaginal portion of the cervix, so that it protrudes outside the vulva, is sometimes spoken of as prolapse, but it should rather be classed under deformities of the uterus.

Really, in speaking of descent of the womb, two main conditions are to be considered:—

(*a*) Descent of the entire uterus.
(*b*) Descent of the cervix only.

A proper understanding of these conditions can only be arrived at by consideration of pelvic anatomy, and more particularly of the pressure relations of the abdomen and its retentive power.[1] In speaking of minor displacements, this was partly considered, and the question of the weight of the uterus in relation to descent discussed, and no more is necessary here than to emphasise the points that the uterus can have no weight in itself, and that the normal pressure relations of the abdomen are, and should be, equal and opposite; and further, that the uterus maintains its normal level by virtue of this balance between the structures that sustain it and the forces that tend to depress it. The so-called ligaments of the uterus have no value in preventing its descent; in fact, it is a pity they are called 'ligaments' at all. Their function seems not to prevent the womb moving, but to give it unlimited movement; they stretch and give to any extent, if a due amount of time be allowed.

There is a certain amount of physiological descent in all women with respiration or effort; the slighter forms of prolapse are exaggerations of this physiological yielding without necessary alteration of relation of parts. If the condition goes further, the descent becomes pathological, for either the uterus sinks into the vagina, inverting the upper part of the vagina, or the anterior vaginal wall sinks down, and pulls the uterus after it.

Generally, the anterior vaginal wall comes down first; this drags down the cervix, and this again pulls down the posterior vaginal wall. The anterior portion invariably contains more or less of the bladder wall (**Cystocele**), owing to the intimate anatomical adhesion of anterior vaginal wall and bladder; a sound in the bladder easily demonstrates this fact. In most cases the whole of the anterior vaginal wall comes down, obliterating the fornix. Then follows the cervix, and then the posterior vaginal wall more or less completely everted; but as a rule, if the perineum be complete, the small portion which is attached to this remains in its place.

This swelling is sometimes called a **Rectocele**, but a sharp distinction must be made between mere prolapse of the posterior vaginal wall, and prolapse of the posterior wall, which drags with it a pouch of the rectum; this can easily be ascertained by introducing the finger into the bowel. In the latter case, the pouching of the bowel forwards may lead to the greatest distress and discomfort. When the bowels act, fæcal matter passes into the pouch, and sometimes the patient cannot get the bowels to act till this same pouch is pushed up. This is a true Rectocele.

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[1] Matthews Duncan, *op. cit.* p. 148.

In rare cases, in large prolapse, the whole of Douglas's pouch is contained in the posterior swelling, and the prolapsed mass may even contain intestine. It is also very rare to get a cystocele without descent of the uterus as well.

The perineum plays no part in supporting the uterus; it merely prevents it for a time from escaping at the vulval outlet. If the perineum be ruptured, or very lax, the event is merely facilitated, and the womb at once comes outside; it is comparable to the delay of the head in child-birth by the perineum.

The essential **cause of descent** is a weakening of the supports of the uterus,[1] these supports being the components of the pelvic floor, and whatever may be the cause of such yielding, the womb cannot come down unless the floor of the pelvis descends too. In fact, the condition is a **hernia**, and there are often associated with it other hernial conditions, such as varicose veins, prolapse of the bowel, piles, or even other definite herniæ, such as femoral or umbilical.

The main causes of yielding of the pelvic floor are:

a. **Weakness**; this occurs with lowered muscular tone.

b. **Injury at Childbirth**, especially if repeated. Multiparity is a very common factor; nearly all cases occur in women who have had children; in such cases there has been injury to the pelvic floor, the levator ani, and its fasciæ. It seems to be a matter of overstretching, rather than distinct injury to muscle fibres or planes of fasciæ, and once having occurred, the changes remain. No real laceration of muscle has ever been dissected,[2] but absorption of fat from cellular tissue of the pelvis has been noted.

c. **Overstretching**, beyond the facts mentioned above, also occurs from overwork in women who have to undertake laborious occupations, or where the abdominal pressure from above is increased by the extra strain, as in cough, constipation, or full bladder.

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[1] Herman, *Diseases of Women*, p. 104.

[2] Herman, *op. cit.* p. 105.

Sometimes this pressure from above may be due to distension of the abdomen by fluid, or some tumour, such as an ovarian, where there is no room for the uterus. The author has seen a case where the whole of the uterus andvagina were pushed down outside the vulva by an ovarian tumour in an old woman ; when this was removed, the uterus could easily be replaced, and remained in good position.

It is very rare to get a uterus prolapsed by the dragging of a tumour from below,[1] *e.g.* a fibroid attached to the fundus or cervix, but cases do occur ; it is commoner for tumours growing from the fundus to produce inversion.

d. **Congenital Defects** are sometimes seen where the uterus is prolapsed in virgins, the vaginal walls being abnormally attached ; in other cases the vaginal cervix is abnormally long and protrudes from the vulval orifice. The condition will be described in the next chapter, but should be considered a congenital uterine defect.

Degrees and Varieties of Prolapse.—(*a*) The cervix may be at or beyond the vulval orifice, while the body of the uterus remains more or less at its natural level. In such cases some part of the uterus has undergone some lengthening, and it is either the vaginal or supra-vaginal portion of the cervix which is affected. Elongation of the pars intermedia is very rare.

(*b*) The whole uterus, unaltered in length, descends, and may be completely external ; it is generally in a position of retroversion, and the vaginal fornices have descended more or less completely, forming a bag, at the bottom of which is the uterus. This is known as simple, complete, or frank prolapse. It is not so common as supra-vaginal elongation of the cervix.

In most of these cases, it is not the mere descent of the womb which is the important factor ; sometimes it appears to be the womb which is the only organ that does not descend. The extreme laxity of everything forming the pelvic floor, the distensibility of the vagina, the loss of tone of its walls, the descent of the bladder and rectum, and the gaping vulval outlet are all important. Many of these changes are traceable to childbirth, to a puerperal period which has been ill-managed, or where the lying-in has been too short.

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[1] See Herman, *op. cit.* p. 115, fig. 25.

Description of particular Varieties.—(a) *Elongation of the Supra-vaginal Cervix.* In this variety the cervix appears at the vulval orifice, the body of the uterus remains more or less in its natural position, and the supra-vaginal cervix is much elongated, at times measuring as much as two inches. This stretching is probably due to tensile elongation combined with a certain amount of hypertrophy ; it is improbable that such a condition is congenital. Invariably the bladder descends, and probably the cystocele is the primary factor ; this pulls upon the cervix at its attachment and drags upon it, and this again brings down the posterior vaginal wall. Both the vaginal fornices are obliterated, and are low down, the anterior vaginal wall being completely descended, and the posterior also, as far as its attachment to the perineum. Some authors think that descent of the uterus is the primary factor.

That there is tensile elongation is evident, for on replacing such a uterus the canal, which before measured four or five inches by the sound, only measures a little over its normal length, and becomes shorter still if the woman be kept at rest in bed for a few days with the uterus well supported.

The long, thinned-out supra-vaginal portion can easily be felt in such a prolapse, like a cord stretching up into the pelvis, about the thickness of one's finger.

The reason why the fundus remains at its normal level is somewhat obscure ; possibly its own attachments keep it there, or rather the attachments of the upper portions of the supra-vaginal cervix, such as the utero-sacral folds. The ordinary so-called ligaments of the uterus cannot have much effect, if any. The body of the uterus in these cases is generally retroverted if not descended, and retroversion is always a precursor of descent.

In supra-vaginal prolapse there is generally great swelling and eversion (ectropion) of the vaginal cervix, the mucous lining of the canal being exposed, and erosion and catarrh are common ; there is much discharge ; while, from its dependent position, there is frequently œdema and swelling. This soon improves, if the cervix be returned and kept within the vagina.

The catarrh and discharge from this everted swollen vaginal portion and the exposed cervical canal are sometimes excessive, and such a cervix looks very ugly, resembling cancer, and yet cancer is rare in such cases.[1] It is often said that such a cervix is ulcerated, but true ulceration of any cervix apart from cancer is very rare, yet in some of these prolapsed cervices, or at all events over the surface of the exposed vaginal walls, true ulcers do form, with a well defined-edge and a red callous or sloughing base. They are painless, and do not bleed readily. On section they are true simple ulcers; they heal at one part and extend slowly at another. If the everted vagina be replaced and kept so, they soon heal, leaving whitish scars. They exactly resemble varicose ulcers of the leg, and are due to malnutrition and œdema of the dependent exposed surfaces.

Friction has little to do with such ulcers, for they occur in positions where there is no friction; nor does the passage of urine over the affected parts cause them, though it may aid in macerating the epithelium. Erosion (which is an adenoma) must be carefully distinguished from true ulceration.

Very similar changes affect the exposed vaginal surfaces in prolapse; at first there is catarrh and discharge and the rugæ are still to be seen, but after constant exposure the surfaces cease to become moist, and are smooth, horny, and dry.

Ulceration of an exactly similar nature takes place here, and the clean, sharply-defined shallow ulcers can be seen healing at one place and extending at others. Or again, the areas may be seen covered with scar tissue.

There is frequently great hypertrophy and œdema. Elongation of the supra-vaginal cervix occurs invariably in multiparæ.

(b) *Elongation of the Pars Intermedia* is very rare, and is only to be mentioned; the pars intermedia being a somewhat fanciful segment of the cervix limited by vaginal attachments of the anterior and posterior walls. Since the posterior attachment lies at a higher level than the anterior, lines drawn across the cervix at each level would include a small portion between them, which is called the pars intermedia, the vaginal cervix being below this part, and the supra-vaginal above. It is said that in this condition, if the pars intermedia be elongated, producing prolapse, the anterior fornix only is descended, the position of the uterus is normal, and the length of the uterus is increased. The patient is always a multipara.

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[1] Sir J. Williams, *Cancer of the Uterus*, 1888, p. 81.

(c) *Elongation of the Vaginal Portion of the Cervix.*—This, as has been previously explained, is really not a prolapse, but a congenital deformity in which the vaginal portion of the cervix is very much elongated, and may protrude at the vaginal orifice. In these cases there is no alteration of the position of the body of the uterus, which is normal and anteverted, neither of the vaginal fornices descends, nor is there any alteration of the relations of the bladder. The length of the uterus is much increased, the sound passing as much as five inches in some cases. The patient is a nullipara.

Some authorities have pointed out that the condition is rather one of high implantation of the vaginal fornices than elongation of the vaginal portion. This seems rather to be borne out in practice, for if the elongated portion be amputated at the level of the fornices, very little of the body of the uterus is left behind.

Sections of the elongated cervix in this form of prolapse show very little departure from the normal tissue; it appears not to be inflammatory, as there is no parenchymatous infiltration.

(d) *Simple, Complete, or Frank Prolapse.*—In this condition the inverted vagina forms a bag hanging from the vulva, at the bottom of which lies the uterus, generally retroverted, otherwise unaltered; it is unchanged, unstretched, and is completely and 'frankly' prolapsed.

Both fornices are descended, often the everted vagina forming a large surface which may by exposure become dry, scaly, cracked, or ulcerated, as before described. The patient is in all cases a multipara. In the posterior pouch of the vagina, besides the uterus, intestine is sometimes prolapsed, but it is rare.

The following table (Champneys) is of use in comparing the various forms of prolapse:—

Name	Descent of Fornices	Uterine Position	Length	Parity
Complete	Both	Retroversion	Normal	Multipara
Supra-vaginal	Both	Retroversion, not necessarily descent	$4\frac{1}{2}$ in.	Multipara
Vaginal	Neither	Normal	$4\frac{1}{2}$ in.	Nullipara
Pars intermedia	Anterior	Normal	Increased	Multipara

Though Cystocele has not been described apart from prolapse, it is the commonest thing which descends at the vulval orifice, and, as before mentioned, the bladder invariably accompanies the anterior vaginal wall. It rarely causes more than mechanical inconvenience; sometimes urine decomposes in the pouch, and at times patients find difficulty in emptying the bladder till they push the cystocele up, as the urethra is displaced; it may be a troublesome complication in labour. The ureters are sometimes dragged upon. It is extremely rare for the vagina to descend alone.[1]

Prolapse of any portion of the uterus is rare with pregnancy. It may occur at the early stages, but as the uterus enlarges and rises into the abdomen the prolapse disappears; later, when the uterus is above the brim, prolapse of the body is impossible. The only cases recorded appear to have been descent of the enormously hypertrophied and elongated cervix; this may give rise to trouble at the time of labour owing to delay in dilatation, and, in the congenital vaginal form, be an insuperable bar to delivery without operative interference.

At the menopause the natural retrograde changes in the uterus may lead to a cure of the prolapse, but it is not necessarily so at all, many very old women having very serious descent of the uterus.

RETROVERSION OF THE GRAVID UTERUS[2]

This is a very important displacement of the uterus, and necessarily ranks as a major one, but the subject rather belongs to midwifery than gynæcology, hence it will only be mentioned here.

It is a very serious displacement, and has serious results. It is one of the commonest causes of retention of urine in women, and such retention may be accompanied with overflow, a fact which should never be overlooked.

Varieties.—(*a*) Sudden retroversion of the gravid uterus about the fourth month, the womb falling backwards, generally by some effort or accident, the fundus becoming jammed behind the promontory of the sacrum.

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[1] Kelly, *Johns Hopkins Hospital Reports*, vol. iii. p. 311.

[2] Matthews Duncan, *Clin. Lect.* 4th ed. p. 400.

(*b*) The uterus, being already in a position of retroversion, becomes pregnant in that position. It enlarges till such a time, generally about the fourth month, when it fills the pelvis; it should then rise to become an abdominal organ. At this period any further disturbance, such as a full bladder, may complete a vicious circle of pressure relations, the uterus reacting on the bladder, and the bladder, becoming fuller, reacting upon the uterus. The retroversion is thus increased, and the uterine circulation impeded. The uterus becomes swollen and engorged, which makes things worse. Eventually, the pressure on the urethra and neck of bladder causes obstruction to the outflow of urine, and retention results; finally, the bladder becomes overful and the urine dribbles away, the bladder all the time being over-distended. This condition of incontinence with overflow must not be forgotten, and a catheter soon decides the condition.

Dangers.—There is possibly nothing so easy to diagnose, and yet retroversion of the gravid uterus is overlooked, generally by carelessness (from which most errors occur), and it may lead to the gravest dangers. The retention of urine in itself is serious if not relieved, and cystitis may supervene upon the use of dirty catheters with grave results, both upon the bladder itself, and upon the ureters and kidneys. The cystitis may be very acute, leading to the sloughing of the bladder[1] or exfoliation of the mucous membrane, either in shreds or more or less *en masse*, with resulting cicatricial contraction of the bladder. Cases have been reported where fistulæ have formed, either vesico-vaginal or vesico-urethro-vaginal, with endless suffering; the bladder has even sloughed through the anterior abdominal wall, and urine has escaped in this way.[2]

The dangers to the urinary tract above the bladder are equally severe; acute nephritis, pyelo-nephritis, and abscess of the kidney may supervene.

With regard to the uterus itself, if the condition be left unrelieved, abortion and its attendant dangers may result, or the uterus may itself inflame and slough. Serious results may ensue from attempts either to empty the uterus, or in its reposition. Retroversion of the gravid uterus may, if not properly treated, become a grave condition. Increased secretion of urine sometimes follows the relief afforded by replacing the uterus.

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[1] Matthews Duncan, *Clin. Lect.* 4th ed. p. 400.

[2] *Op. cit.* p. 404.

Retroversion of a uterus enlarged by fibroids may exactly resemble the above condition and lead to similar serious results. It is pelvic fibroids which give rise to so much trouble, not the large abdominal masses above the brim. A fibroid uterus of the size of the fourth month gravid uterus so nearly fits the brim, or being above the brim so easily gets impacted, that any small disturbance of pelvic pressure relations, such as a full rectum or bladder, may lead to such a tumour becoming retroverted or jammed in the pelvis, and the most disastrous pressure results ensue. More will be said of this in the chapter on Uterine Fibroids.

The **retroversion of the gravid uterus as described by Oldham** is a different matter; it is not a retroversion at all, but more probably a curious pouching of the posterior wall of the lower uterine segment, which is held down in the pelvic excavation by adhesions in Douglas's pouch,[1] so that at full time the part of the fœtus that occupies this pouch is below the brim even long before the onset of labour, the cervix being high up, close to the symphysis.

Pendulous Belly.—This condition, described by Matthews Duncan,[2] is the extreme anteversion of the uterus in advanced pregnancy, the uterus falling through, or distending extremely the linea alba, and stretching the peritoneum and skin between the divaricated recti; sometimes so great is the anteversion that the pregnant uterus may reach the patient's knees.

It may be more or less a simple matter, but it leads to the suspicion of contracted pelvis, as the fœtus cannot enter the brim, and all such cases should be at once measured. Apart from pelvic contraction, anterior obliquity of the uterus leads to delay, the forces of labour acting in a wrong direction; but if the uterus be replaced, and retained by a binder more or less in the normal position, labour may go well.

Matthews Duncan[3] describes a case of anteflexion in a primipara which could not be replaced as in the common pendulous belly; it was really not displaced secondarily, but grew into this peculiar shape and position. Matthews Duncan, speaking of pregnancy and displacements, says: 'When a woman with a displaced uterus becomes pregnant, it may assume early what is called a normal position, or a woman becoming pregnant with the womb in a normal position may soon have it displaced, or she may become pregnant with the uterus displaced, the organ remaining so during the early months.'

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[1] Matthews Duncan, *Clin. Lect.* p. 401.

[2] *Op. cit.* p. 401.

[3] *Clin. Lect.* 4th ed. p. 401.

Inversion of the Uterus.[1]—Inversion of the uterus is possible at any time, but in its acute form it is an accident of delivery, and is one of the most interesting and important, as well as the most rare, in midwifery. It would be out of place to go into this matter fully in a book on diseases of women, but mention must be made of it in its relation to chronic inversion.

Acute inversion of the uterus occurs in the third stage of labour, and its primary factor is always inertia, complete or partial.

It is generally attended with the gravest symptoms, often with death; other cases pass unnoticed and become chronic, with little disturbance of the general health. As was said in a former chapter on minor displacements, these affections, paltry as they are, are supposed to give rise to such tremendous results, immediate and remote, and yet a uterus turned inside out may give rise to none, or to very few symptoms at all.

Acute Inversion may be:—

- *a.* Spontaneous: that is, the woman produces it herself.
- *b.* Artificial: the woman produces it with the aid of an attendant.
- *c.* Passive: *i.e.* the whole uterus is relaxed (complete inertia).
- *d.* Active: this means the final stage of inversion is completed by uterine contractions.

Matthews Duncan describes four varieties of acute inversion:[2]

1. Spontaneous passive; 2. Artificial passive; 3. Spontaneous active; 4. Artificial active.

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[1] Matthews Duncan, *Clin. Lect.* p. 424.

[2] *Id.*, 'Contribution to the Mechanism of Natural and Morbid Parturition,' *Obstet. Soc. Edin.* 1875, p. 277.

These may be best explained by the following table (Champneys), 'St. Bartholomew's Hospital Journal,' 1896, and 'Clinical Lectures' :—

Name	Predisposing cause. State of Uterus	Exciting cause	Attendant
1. Spontaneous passive.	All relaxed	Bearing down	Not to blame
2. Artificial passive	All relaxed	1. Traction on cord 2. Unskilful pressure from above	To blame
3. Spontaneous active	Placental site relaxed	1. Bearing down, producing introversion 2. Expulsion of introverted part by uterine contractions	Not to blame
4. Artificial active	Placental site relaxed	1. Traction on cord, producing retroversion 2. *a*. Unskilful pressure from above, producing introversion *b*. Expulsion of introverted part by uterine contractions.	To blame

Degrees of Inversion.—These may be :

a. A portion of fundus only.

b. Introversion, the whole fundus turned into the cavity of the uterus, but not through the cervix.

c. Complete inversion, the common condition, at all events in the acute cases. In this, the whole of the uterus is inside out, except the cervix, which is not commonly affected. In fact, so different are the body and cervix in their behaviour and structure that it would be almost better to describe them as more or less separate structures. This is especially seen in labour. So, in inversion, inertia affects the body chiefly, and inversion may really be termed a disease of the body only.

d. Inversion of one wall. This is rare, but it possibly occurs as the primary factor in acute inversion where the placental site is the passive portion of the uterus, and the rest of the uterus more or less active. It may occur in chronic inversion, owing to the traction of fibroids, or at the attachment of a mass of malignant disease.

Chronic Inversion.—This is said to be the sequel of acute, but since a chronic disease should own a chronic cause, the logic is bad, equally so the generally accepted definition that acute inversion becomes chronic when the period of involution is complete. Still, there is no doubt that some cases of chronic inversion which must have originated in the third stage of labour have passed unnoticed during the puerperium, and perhaps years later the patients show the symptoms and signs of inversion which is now certainly chronic.

Another set of cases of chronic inversion do from the first own a chronic cause: Fibroids, which, in their expulsion from the uterine cavity by active contractions of the uterus itself, may drag with them the base to which they are attached, and if at the fundus, or on one wall, will eventually cause chronic inversion, which in this case is certainly spontaneous. Sessile submucous fibroids seem to cause chronic inversion oftener than those that are polypoid.[1]

Chronic inversion may also be caused by cancer of the body of the uterus.[2] Sarcoma [3] produces this effect more often.

In removing fibroid polypi where strong traction is made on the pedicle, it is well to remember that there may be partial inversion as well, else an awkward accident may occur.

Involution, *i.e.* the natural physiological retrograde process after delivery, goes on perfectly well even when a uterus is inverted, which is a very remarkable fact, considering that the whole organ is turned more or less inside out. Involution is generally complete in six to eight weeks.

In *Chronic Inversion*, the uterus is more or less turned inside out, the body being inverted into the uterine cavity, and it may or may not protrude beyond the cervix. Generally, as before mentioned, the cervix itself does not share in the inversion, but it may do so, if the fundus be pulled upon.

The uterus itself is correspondingly shortened, and a sound only enters a short distance by the sides of the inverted mass. This is a great point in the diagnosis from fibroid polypi, where the length of the uterus is generally increased.

[1] Matthews Duncan, *op. cit.* p. 310, says: 'It is not the polypoid form which produces this effect, but a sessile or necked tumour, the uterus seizing the tumour as it seizes a child, and pushing it out.' See also Herman, *Trans. Obstet. Soc. Lond.* vol. xxx. 1888, p. 226.

[2] H. Kelly, *Operative Gynæcology*, p. 536.

[3] Targett, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 285.

The tubes and portions of the broad ligaments are drawn into the hollow of the inverted fundus above, and a cup-shaped depression is produced which it is said can sometimes be actually felt on bimanual examination.

The most astonishing feature of this cupping and drawing in of the tubes is that, even after chronic inversion has existed for years, no adhesion whatever may take place within the cup, and the uterus can always be replaced by proper methods. Such capability of replacement is mainly due to elasticity, distensibility, and muscular relaxation of the whole uterus, not relaxation of any constriction of the cervix or any other particular portion of the uterus; so, in treatment, reposition is obtained by a small force long applied till the whole uterus relaxes and yields. Much has been said of this constriction of the cervix in such cases, and that it may cause ulceration and sloughing of the inverted fundus; an inverted fundus has been known to die and slough off, but it is not due to the constriction of the cervix; such constriction has never been seen or described.[1] A fibroid polypus occasionally sloughs off when its stalk passes through the cervix, yet it is not tightly constricted by it, nor even more firmly constricted than when the polypus was alive.

Generally such cases of sloughing are due to septic infection and disintegration, often started by some injury or attempts at removal.

Cases are on record where, instead of sloughing, spontaneous replacement of an inverted uterus has occurred.

In chronic inversion a rounded, pear-shaped, smooth mass is found occupying the vagina, encircled by the cervix uteri, but not constricted. The mass is not tender, though sensitive, and does not bleed unless roughly handled. The openings of the Fallopian tubes may be very rarely seen on the inverted mass, and the whole length of the uterus is diminished, as demonstrated by the use of the uterine sound.

The symptoms of chronic inversion are achings, leucorrhœal or other discharges, and loss of blood; these may vary in persistence and amount. If inversion be caused by a fibroid, this may give rise to great and very serious hæmorrhage.[2]

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[1] Matthews Duncan, *ibid.* p. 26.

[2] Matthews Duncan, *Clin. Lect.* p. 425.

CHAPTER VIII

ON IMPERFECT DEVELOPMENT AND MALFORMATION OF THE UTERUS

The development of the uterus may be imperfect in many ways. Some defects prevent uterine function, others do not. Some producing amenorrhœa have already been discussed.

Age changes[1] must not be confounded with imperfect development. The uterus undergoes important changes between birth and puberty. In the new-born infant the uterus has no fundus, its summit may be notched, the cervix is very large in comparison to the body, and the arbor vitæ is distinct. In the child the uterus is an abdominal, not a pelvic organ, the body of the uterus lying above the level of the brim, its anterior surface curving forward towards the bladder.

Towards puberty, the fundus becomes more and more marked and more developed, till it assumes its mature characters. After the menopause, it shares in the general atrophy of the reproductive organs, and the cervix becomes less and less marked, and no longer projects into the vagina, and in very old women no cervix can be felt, but only the dimple of the external os.

Malformations of the Uterus may be classified[2] as:

a. Those due to defect of growth.

b. Those due to imperfect union of the lateral halves.

During the first eight weeks of fœtal life the ducts of Müller are separate; at the end of the twelfth week the septum is absorbed; by the end of the twentieth week the horned appearance of the uterus is lost, the arbor vitæ is formed, but the vagina is still smooth; at birth the vagina is rugose and the uterus rounded, having the shape previously described.

Many of the anomalies of development of the uterus and vagina are due to irregularities in the fusion of the lower part of the Müllerian ducts, in their mode of termination, their partial or complete absence, and their imperforate condition. Abnormalities of the vagina have already been considered. It is possible perhaps to classify uterine deformities according to the period of intra-uterine life in which they are produced. L. Fürst, quoted by Ballantyne[1] in the latter writer's article on Malformations of the Genital Organs (*q.v.*), divides them into :

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[1] Bland-Sutton and Giles, *Diseases of Women*, p. 126.

[2] Herman, *Diseases of Women*, p. 605.

1. **Those originating between the first and eighth week.**—Partial or total absence of the uterus, rudimentary uterus, or absence or deficiency of one half, want of union of the lateral halves, the cavities being completely separate or only partially so.

2. **Those originating between the eighth and twentieth week,** such as certain minor malformations characterised by trifling alterations in form, and by the presence of a more or less marked septum internally.

3. **Originating between the twentieth and fortieth weeks.**—In this, the uterus retains its fœtal characters, in the disproportionate size of the cervix to the body.

Ballantyne[2] himself thinks this scheme too complicated, and proposes the following three groups :—

- *a.* Those in which there is apparent excessive formation, *i.e.* doubling.
- *b.* Those in which defect is the leading character.
- *c.* Those which show altered relationship of parts.

Practically **all the important deformities of the uterus** are produced by influence acting in the first half of pregnancy. They are[3] :—

1. **Absence of the Uterus.**
2. **Rudimentary Uterus.**
3. **Absence or deficiency of one half.**
4. **Want of union of the lateral halves,** which, according to its degree, may result in either
    - *a.* A completely double uterine cavity, the two halves being
        1. Quite separate = uterus didelphys.
        2. To external appearances united, divided only above = uterus bicornis.

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[1] J. W. Ballantyne, article, 'Malformations of Genital Organs in Women,' with complete references; Allbutt and Playfair, *System of Gynæcology*, p. 73.

[2] Ballantyne, *op. cit.* p. 74.

[3] Herman, *Diseases of Women*, p. 605.

    3. Of normal external appearance, but divided by a septum = uterus septus.
  b. An incompletely divided uterine cavity, the uterus being either
    1. Horned = uterus bicornis or arcuatus.
    2. Not horned = uterus subseptus.

Influences acting in the second half of pregnancy only produce the fœtal or infantile uterus. (Herman.)

1. **Absence of the Uterus.**—This, in the adult and otherwise normal individual, is very rare indeed,[1] and probably without complete post-mortem examination it cannot be certainly said to occur; in some cases so described the individual was a male with undescended testes. Absence of the uterus is found in fœtal monstrosities, such as the *acardiac* and *sympodial* varieties. When the Fallopian tubes as well as the uterus are absent, the peritoneum passes directly from the bladder to the rectum. If the tubes be present, it forms a sort of mesentery for each,[2] the round ligaments may be sometimes seen ending in some cellular tissue between the bladder and the rectum. The ovaries are very rarely normal, the tubes generally not perforate, and the vagina wanting in most cases. Amenorrhœa is practically constant, but ovulation may occur.

2. **Rudimentary Uterus.**—This term is generally meant to include cases where, in place of a normal uterus, a body of variable form is discovered consisting of fibrous, muscular or fibro-muscular tissue, sometimes solid, at other times having a rudimentary cavity. The small solid mass of muscular tissue is found in the middle line, between the folds of the broad ligament, which seems to reach right across the pelvis in one fold. The tubes, ovaries, cervix, and vagina are absent, or imperfect, but the external genitals are generally well-formed, though the pubic hair may be scanty, and the breasts small.

The most common form of rudimentary uterus is the *Uterus bi-partitus*,[3] in which the body of the uterus is very small and formed of fibro-muscular tissue, ending below in a blind vaginal cul-de-sac, into the top of which the fibro-muscular bundles are inserted; above it ends on each side in a fibro-muscular band, which is sometimes swollen at the insertion of the round ligaments and may even contain a small cavity;

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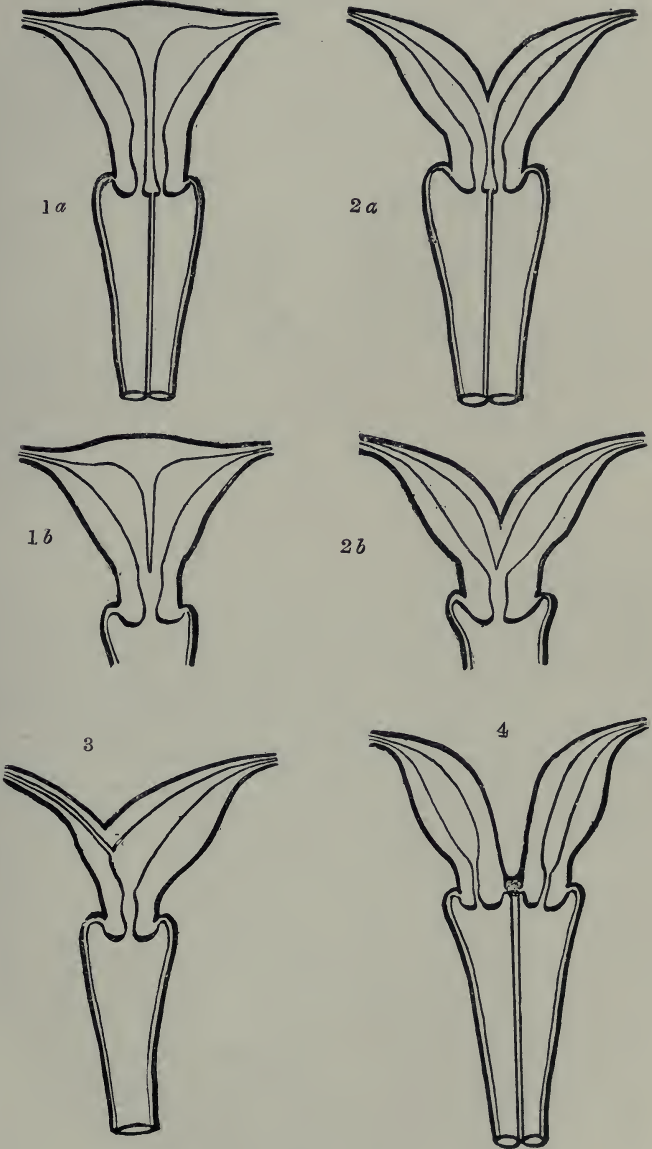
[1] Ballantyne, *op. cit.* p. 81.

[2] Ballantyne, p. 81.

[3] Herman, *Diseases of Women*, p. 607.

Fig. 22.

DIAGRAM TO ILLUSTRATE THE VARIETIES OF DOUBLE UTERUS.



1 *a*. Uterus septus.
1 *b*. Uterus subseptus.
2 *a*. Uterus bicornis.
2 *b*. Uterus bicornis unicollis (semi-duplex)
3. Uterus unicornis.
4. Uterus didelphys.

(Giles, 'Trans. Obstet. Soc. Lond.' vol. xxxvii. p. 304.)

or the neck may contain a cavity and the rudimentary body be solid. The vagina may be short or absent.

It is impossible to distinguish clinically between absence and a rudimentary condition of the uterus, but the existence of one or other can be made out by careful rectal, vaginal, and bimanual examination. With the help of the bladder sound nothing like a well-formed uterus can be found between the rectum and bladder; the transverse band between the tubes can sometimes be felt like a sling, between the sides of the pelvis; the ovaries may, or may not be, palpated. It is important to define these conditions with precision, for when they exist no treatment, medical or surgical, can ever do any good.

3. Malformations in which there is absence or deficiency of one half of the Uterus.

Uterus Unicornis. (Fig. 22, No. 3.)—In this condition one horn alone is well-formed. There are two varieties:[1]

(*a*) That in which the second horn is altogether absent.

(*b*) That in which there is a solid or hollow rudiment of the second horn.

In the former case there is complete defect in one of the Müllerian ducts. The uterus unicornis has really no fundus; the single horn inclines to one side of the middle line, tapering to a point at which it is continuous with the Fallopian tube; here the round ligament is attached. The body of the uterus is smaller than when that organ is properly formed, and the neck appears longer and thicker than the body; the vagina is narrow, absent, or septate. On the opposite side,[2] if the other half uterus is absent, then tube, ovary, and round ligament are wanting too; if imperfectly developed, these parts are undeveloped. With this condition the appendages have been found apparently unconnected with the uterus, lying in the lumbar region; sometimes the corresponding ureter and kidney may be wanting, and the bladder only developed on one side.

If the rudiment of the second horn be present, it may be solid or hollow, and, in the latter case, the cavity may or may not communicate with that in the first horn. This rudimentary horn may be the seat of a pregnancy,[3] or a collection of menstrual blood may be found in it. With relative frequency either the rudimentary or the well-developed horn may become the seat of a fibroid tumour.[1]

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[1] Ballantyne, *op. cit.* p. 79; see also Herman, *op. cit.* p. 607, 609.

[2] Herman, *op. cit.* p. 607.

[3] Ballantyne, *op. cit.* p. 80.

There may be amenorrhœa with uterus unicornis, or menstruation may be normal. Rudimentary horn pregnancy is described in the chapter on Extra-uterine Gestation ; there is a very good specimen [2] in St. Bartholomew's Hospital Museum. Rupture eventually takes place, with severe intra-peritoneal hæmorrhage; it is a highly dangerous condition. Howard Kelly [3] gives us a very good drawing of such a case, where it will be noted how low down near the cervix the rudimentary horn is attached, while on the opposite side the appendages come off from the top of the developed horn. In such cases the attachment of the round ligament settles which is tube and which is uterus. (See Fig. 56.)[4]

Sometimes the presence of a uterus unicornis may never be discovered during life, or only at an abdominal section for something else. Even normal delivery may occur from the well-formed horn and the condition remain unnoticed, although in pregnancy it may be suspected from the obliquity and slenderness of the uterus.[5] Pregnancy in the rudimentary horn cannot be generally diagnosed from a tubal pregnancy.[6]

4. Want of union of the lateral halves of the Uterus, the two halves being quite separate.

Uterus Didelphys.[7]—In this form both uteri are quite distinct, viscera sometimes lying between them. The condition is frequently combined with other malformations, and such children are generally still-born. The two halves of the uterus have remained externally distinct and can be moved independently of one another; they are of great interest pathologically, for Hæmatometra [8] and Pyometra have occurred in one half of such a uterus. Gestation has been known to take place in one half, or in both halves simultaneously;[1] other cases in adults have been described uncomplicated by gestation or restrained menstrual products, or have been merely discovered accidentally on the post-mortem table.[2]

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[1] Doran, 'Fibroid in Uterus Unicornis,' *Brit. Med. Journ.*, vol. i. 1899, p. 1389, with reference.

[2] Museum, St. Bartholomew's Hospital, No. 3078. See Fig. 57.

[3] H. Kelly, *Operative Gynæcology*, vol. ii. p. 465.

[4] See also fig. 55, p. 146, in Taylor's work on *Extra-uterine Pregnancy.*

[5] Herman, *op. cit.* p. 609.

[6] See, however, Targett, *Trans. Obstet. Soc. Lond.* vol. xlii. Oct. 1900.

[7] Giles, *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 301, with literature ; Kussmaul, *Verdoppelung der Gebärmutter*, Würzburg, 1859, p. 107 ; Müller, *Der Sterilität der Ehe*, 1885.

[8] Cullingworth, *Amer. Journ. Obstetrics*, vol. xxvii. 1893 ; *id. Brit. Med. Journ.*, Jan. 6, 13, 1900, and *Trans. Amer. Gyn. Soc.* vol. xviii. 1893, p. 434.

The two halves may menstruate separately or together; if pregnancy occurs in one side, a decidua forms on the other. The vaginæ are invariably separate, though united by connective tissue, and a loose bridge of connective tissue and peritoneum stretches between the cervices.[3] Each uterus has its own Fallopian tube, whose point of junction with the uterine body is indicated by the origin of the round ligament; it has also its own ovary. The two halves are often unequally developed, and one vagina may end blindly above the vulva, so that the corresponding uterus is quite shut off from the outside.

The malformation is due to the non-union of the two primitive Müllerian ducts which should fuse during the third month of fœtal life.[4] Variations in degree of this want of fusion also produce the uterus septus or bicornis. Besides cases occurring in still-born children, Giles[5] refers to authentic cases occurring in adults.

The clinical complications[6] to which this malformation may give rise are, obstruction to labour from the displaced empty half, obstruction due to a vaginal septum, retained and undiscovered products of conception in one half in cases of double pregnancy, double vaginitis or endometritis (treated unsuccessfully by therapeutical applications on one side only), dyspareunia, and unilateral atresia, with retained menstrual products. One of Galabin's cases is of interest, as a fibroid grew from one side of a uterus didelphys.

Uterus bicornis. (Fig. 22, No. 2 *a*.)—This is a commoner malformation than uterus didelphys; here the two halves or horns are not entirely separate, but united more or less intimately at their lower end, in the region of the cervix. Müller's ducts have begun to fuse, but have stopped short of the normal, and the uterus is thus two-horned. The uterus bicornis is the connecting link between the uterus didelphys (fig. 22, No. 4), which shows two separate uteri, and the uterus septus (bilocularis) (fig. 22, No. 1 *a*), in which outwardly the organ gives no sign of duplicity.[1] In uterus bicornis the two horns may be of equal size, or one may be much less developed than the other, somewhat like the uterus unicornis, but there are many intermediate varieties.

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[1] Giles, *op. cit.* p. 316. See *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 330.

[2] Galabin, quoted by Giles, *op. cit.* p. 325.

[3] Giles, *op. cit.* p. 305.

[4] Amand Routh, 'Ten Cases of Congenital Malformation of the Uterus and Vagina,' *Illus. Med. News*, March, 1889, p. 224; *id. Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 157.

[5] Giles, *op. cit.* pp. 303, 308.

[6] *Op. cit.* p. 323.

Generally the separation of the two horns is well marked, and between them a ligament or band (recto-vesical ligament) may be found, passing from the bladder to the rectum; sometimes the two horns lie parallel with one another, or may be directed forward, or there may be only the very slightest depression between the two nearly fused cornua. The cervix may show a double orifice (uterus bicornis duplex or bicollis) or may be large and single (uterus bicornis unicollis). (Fig. 22, No. 2 *b*.) The vagina may be double, septate, or single, and the external genitals well formed.

Ballantyne[2] says that there may be anomalies of neighbouring, or more distant organs, for example ectopia vesicæ and polydactyly, or such monstrosities as cyclopia and anencephaly; these generally occur in non-viable infants.

On section, a uterus bicornis shows a well-marked septum between the horns, even in that part which externally appears single. The horns may sometimes be imperforate at one or more places, and menstrual blood may accumulate in one or other horn. Menstruation in other cases may be quite normal or alternate from each horn, or hæmatometra of one side may occur if the cavity does not communicate with the vagina.

Pregnancy may occur in both horns or in one,[3] and if in both, possibly at different dates, perhaps explaining some cases of superfœtation (Ballantyne). Labour may be quite normal, or, if the horn be imperforate, rupture may occur, or the septum between the two horns may be torn through and delivery take place thus. Abortion may also occur.

The sub-varieties[4] of uterus bicornis (also called uterus bicornis duplex) are:

(*a*) **Uterus Semi-duplex** (fig. 22, No. 2 *b*.), in which the two horns join at the os internum, and below the point of junction there is no division at all, or a division not reaching the os externum.

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[1] Ballantyne, Allbutt and Playfair's *System of Gynæcology*, p. 76; Herman, *op. cit.* p. 609.

[2] Ballantyne, *op. cit.* p. 77.

[3] Roberts, 'A Case of Uterus Bicornis accidentally discovered after the Patient's Fourth Pregnancy,' *Brit. Med. Journ.* vol. i. 1900, p. 1282.

[4] Herman, *op. cit.* p. 609.

(*b*) **Uterus Arcuatus.**—This is the slightest degree of all, and there is merely a vertical depression in the middle of the fundus uteri.

Uterus Septus, or Bilocularis. (Fig. 22, 1 *a.*)—In this case the uterus externally shows no sign of division, but is divided internally by a septum into two halves, more or less complete, antero-posteriorly.

Uterus Subseptus or Semi-partitus. (Fig. 22, 1 *b.*)—In this the septum is incomplete; it may start from the fundus and not reach all the way, or be present in the cervix only. It is a less common condition than uterus bicornis. There may be some grooving externally or some extra breadth to indicate the malformation; the vagina may be single, double, or partially septate. Menstruation in a double uterus is generally as perfect as in the normal condition, and, if completely double, sometimes one uterus bleeds, sometimes both.[1] The different varieties of uterus septus show a more complete fusion of Müller's ducts than the uterus bicornis, still it is not quite complete, as shown by the more or less perfect septum which remains. In uterus septus abortion is said to be common; Ruge, quoted by Ballantyne,[2] divided the septum in a case of a patient who had twice aborted, and in the next pregnancy the patient went to term. Malpresentation may occur; the placenta may have dangerous attachments, either low down or to the septum. Pregnancy in one side has ruptured into the other, and if the horn does not communicate with the vagina, unilateral hæmatometra may result in connection with menstruation. Most cases of uterus septus and subseptus are only discovered accidentally.

Malformation occurring in the later months of Fœtal Life, or Childhood.—Only one class of deformity occurs in this group, viz. in the *Fœtal or Infantile uterus.* Here, the anatomical characters which are normal in the uterus in intra-uterine life may persist, and be found in the adult.[3] The uterus of the grown woman here resembles both in size, shape, and thickness of walls, and in relation between the length of cervix and body, either that of a fœtus of six months, or that of a child under fourteen. This latter is sometimes called the Uterus Pubescens; here the organ is small in size, weighs less than normal, and has a cervix and body of practically equal length; the ovaries, tubes, vagina, and mammæ may or may not share in the changes. In the Fœtal or Infantile uterus the cervix is longer than the body and its walls are thick, those of the body being thin; the cervix may be conical and the os externum narrow.[1] The whole uterus is very small and cylindrical, and the sound only passes for an inch or an inch and an half. The endometrium may be poorly developed, and its glands few. The vagina may be short or it may be normal. The breasts are generally small, and the external genitals imperfect; the ovaries and tubes may be normal or rudimentary.

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[1] Herman, *op. cit.* p. 610.

[2] Ballantyne, *op. cit.* p. 79.

[3] Herman. *op. cit.* p. 610.

Sometimes patients are full-grown, sometimes cretinoid,[2] sometimes there is no cavity to the body of the uterus, or its walls are very thin, or the whole uterus may be proportionately small (the Primary Atrophy of Virchow). Herman describes another form, the Uterus Parvicollis or Acollis, in which the vaginal portion is small, but the body normal.

With any of these forms of fœtal uterus, amenorrhœa is common, or, if menstruation occurs, it is attended with pain; sterility is the rule. No treatment in the world will ever make such a uterus grow, and it is not to be attempted.

Uterine Atresia and Stenosis have already been considered in connection with Retention of Menses, and the condition of Hæmatometra and Unilateral Hæmatometra described.

Congenital Prolapse of the Uterus.[3]—This is an extremely rare condition. Ballantyne relates such a case and refers to others, in all of which there was the association of spina bifida. There is real displacement downwards of the whole uterus, as well as a hypertrophic condition of the cervix.[4]

Congenital Elongation of the Vaginal Portion[5] is considered in the section on Descent of the Womb. In this variety the vaginal portion only protrudes at the vulval orifice and neither fornix descends, the body of the uterus itself being in the normal position. It may be possibly a condition of hypertrophy, but is in the author's opinion congenital; it is certainly not due to stretching. Some of the apparent lengthening of the cervix appears to be due to an abnormally high implantation of the vaginal fornices.

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[1] Ballantyne, *op. cit.* p. 82.

[2] Herman, *op. cit.* p. 610.

[3] Ballantyne, *op. cit.* p. 84.

[4] The author had a curious case under his care at the Samaritan Free Hospital, in a young girl of fifteen, in which the whole uterus and vaginal walls were prolapsed outside the vulva; the condition was probably congenital.

[5] Gervis, Allbutt and Playfair, *op. cit.* p. 236.

Abnormal Communications of the Uterus.—In some curious cases the uterus may be connected with the rectum or bladder, or both. Ballantyne[1] mentions a case reported by Doran, in which the right side of a bipartite uterus opened on the outer surface of the body. The uterus has also been found communicating with the ascending colon.

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[1] Ballantyne, *op. cit.* p. 85.

CHAPTER IX

ON STERILITY[1]

To the late Dr. Matthews Duncan we owe very much of our knowledge of this particular subject, as he was the first to apply any scientific methods in its study. Duncan, in his well-known 'Clinical Lectures,' says that 'in lecturing on sterility of women I am embarrassed by dealing mainly with only one-half of the matter, probably the chief or predominant half. I still say probably the chief or predominant half, but enlarged experience and inquiry make me more and more convinced of the greatness of the part played by the male, and that not only in absolute sterility, but in the other allied forms.'

That is to say, that there are two parties to be considered in sterility, not the woman only; and it is unsound treatment to subject a woman to prolonged painful or dangerous treatment for such sterility, unless assured of the potency of the male,[2] who should be examined; and Duncan[3] found that in at least half of the cases of women sterile after treatment for spasmodic dysmenorrhœa the male was at fault.

Sterility may be either absolute or relative.—Absolute Sterility[4] means absolute failure of conception; there is no child, no miscarriage, no abortion; it occurs in about 10 per cent. of married women. A man or a woman may be absolutely sterile in whose anatomical and physiological condition no flaw whatever can be found.[5]

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[1] Matthews Duncan, *Fecundity, Fertility, and Sterility*, 1866; *id. Clin .Lect.* 4th ed. 1889, p. 166. Champneys, 'Sterility,' *Clin. Journ.* Feb. 22, 1893, p. 275; M. Duncan, *Sterility in Women*, 1884; Henry Gervis, article, 'Sterility,' Allbutt and Playfair, *System of Gynæcology*, p. 231; Herman, *Diseases of Women*, p. 630.

[2] Matthews Duncan, *Clin. Lect.* 4th ed. p. 167.

[3] *Ibid.*

[4] Herman, *Diseases of Women*, p. 630.

[5] Matthews Duncan, *Clin. Lect.* p. 168.

Relative Sterility is that in which a woman produces children in number not according to her condition, age, and length of married life. It has to do with those conditions giving rise to abortions, infrequent births, and still-born children. In fact, anything which limits the number of children effectively, anything which is prejudicial to the normal number of children produced, is part of the question of relative sterility.

A classification of sterility is one of great difficulty, but the writer proposes to follow that of Champneys in his 'Clinical Lectures' at St. Bartholomew's Hospital.

The **Causes of Sterility** may be considered under two main headings:

(*A*) **Causes** prejudicial to the production, or health, of the sperm or germ cell (**failure of production**).

(*B*) **Causes** prejudicial to the junction of the sperm and germ cell (**failure of exchange**).

Regarding these two classes, it is only failure of exchange that can be considered practically, as the causes of failure of production can seldom be controlled, nor, indeed, can many cases of failure of exchange.

'But the whole of class (*A*) and the whole class (*B*) have to do with a very important part of our duty, *i.e.* prognosis. If the prognosis be hopeless, treatment is useless, and not to be undertaken.'[2]

Now, taking the question of failure of production, the influence of its causes is partly known in plants. Some trees bear no fruit, or if they bear fruit it falls before it is mature; so we may get oranges with no pips, and trees bearing fruit with seeds, and yet these seeds come to nothing; these are failures in the germ and sperm cells in plants. (Champneys.) Again, taking wild animals in confinement: some breed, others do not attempt to breed, others do, and nothing comes of it.

It is a common error to regard the female as the weaker sex; this, in the consideration of reproduction, is also a mistake. Males are much the more delicate of the two, and the laws concerning men are much more subtle and obscure, at all events as regards general health and age. Age in connection with reproduction in man is a thing unknown; there is no 'age of climacteric,' or at least the fall of fecundity and the increase

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[1] Champneys, *Clin. Journ.* Feb. 22, 1893, p. 275.

[2] Champneys, *op. cit.*

of sterility as age advances, is much slower than in woman.[1] In women the greatest cause of sterility is age.

Turning now to the subdivisions:

Class A. Failure of Production.

1. *The Man may be sterile*:

a. **From congenital ill-development and complete absence of semen,** or absence of living spermatozoa in the semen, which condition may be present in a male quite potent as to intercourse.[2]

b. **Bad health.**

c. **Intermarriage** (but hybrids die out).

d. **Want of sexual affinity and incompatibility.**[3]—The man or the woman is capable of breeding, they marry and do not breed; or a man has begotten children by one wife, marries again, and his second wife is sterile, then he dies, his widow remarries and is fertile by her second husband. Incompatibility as a cause of sterility can neither be explained nor cured, nor can its frequency be estimated.[4]

Insanity and its relations are causes of sterility in the male, and bad specimens of the race do not tend to reproduce themselves.

e. **Gonorrhœa** is probably accountable for about one sixth of all cases of sterility in the male.[5] The semen is at fault, and contains pus, blood, or diseased sperm cells.

Sometimes a long history of gonorrhœa, with repeated swelling of the testicle or epididymitis, can be obtained even in a man who is sexually vigorous.

Gonorrhœa has a much more serious bearing on sterility than syphilis. Syphilitic men can breed, and, unfortunately, often do so.

Causes of Sterility in the Female.

1. **Congenital ill-development of organs, especially the ovaries.**—True absence of the ovaries is so rare that its occurrence has been doubted. Small ovaries occurring with amenorrhœa are less rare, but it is not known how small an ovary may be, and yet produce ova capable of development.[6]

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[1] Körösi, quoted by Matthews Duncan, *Clin. Lect.* 4th ed. p. 170.

[2] Gross, *Impotence and Sterility*, p. 87. Male at fault in one case in six, in cases of sterility in marriage.

[3] Matthews Duncan, *Clin. Lect.* 4th ed. p. 168.

[4] Herman, *op. cit.* p. 631.

[5] Gross, *op. cit.*

[6] Herman, *op. cit.* p. 632.

Smallness of the uterus is said to be a cause of sterility; it generally occurs with imperfect development of the ovaries. If the uterus have no cavity, pregnancy cannot take place, but pregnancy may occur in Fallopian tubes, or in a cornu of a uterus so badly developed that its cervical canal cannot be perceived,[1] so that it is rather the *production* of ova that is so essential to fertility.

It is noteworthy, as Champneys [2] points out, that women in whom the ovaries are deficient, and the uterus small, do not have a pelvis of the female type.

2. **Age.**—Duncan [3] has investigated this point with the greatest care, and his tables show that fecundity is greatest [4] in women married between the ages of twenty and twenty-five. 'At this age woman has the lowest risk of sterility, the greatest likelihood of having healthy children that will long survive, the greatest likelihood of herself surviving childbirth, the lowest risk of having abortions, of having excessive family, of having plural pregnancy, and of bringing forth idiots.' [5] Of women married before this age, the earlier they are married the greater the prospect of sterility; of those married after this age, the later they marry, the more likely are they to be sterile. No cause of sterility approaches age in its extent or power.

The age of fecundity, according to Matthews Duncan, generally extends over a period of fifteen years, and he gives it as between the ages of twenty-three and thirty-eight, the pelvis during this period having finished its growth, and the processes of reproduction being carried on with the greatest success.

Further, it must be remembered that the fertile age is not co-extensive with menstruation, the fertile age beginning later and ceasing earlier.

3. **Defects in the General Health.**—Chlorosis and obesity are factors against fecundity. Very fat women do not breed, and when menstruation ceases a woman often gets very fat. There is a marked antagonism between growth and genesis.[6]

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[1] Herman, *op. cit.* p. 632.

[2] Champneys, *op. cit.*

[3] Matthews Duncan, *Clin. Lect.* 4th ed. p. 169.

[4] Herman, *op. cit.* p. 632, who gives Duncan's tables on p. 631.

[5] Matthews Duncan, *Clin. Lect.* p. 169.

[6] Champneys, *Clin. Lect.* and *Clin. Journ.* Feb. 22, 1893.

4. **Want of sexual affinity** has already been considered in the male. As regards the female, the same may hold good, and yet is equally inexplicable.

5. **Inflammation of or around the ovaries.**—This has already been considered from a pathological point; in connection with sterility, it may be prejudicial to the escape of the germ cell, but even in a woman who has had inflammation of her ovaries it would be very unwise to say that she can never become pregnant. A woman who has had a very bad labour may still have other children. 'Pelvic peritonitis makes pregnancy less likely, and inflammation in the pelvis sometimes makes the sexual function a cause of suffering, but these are temporary, not invariable and permanent effects.'[1] The amount of unchanged ovary necessary for reproduction is very small indeed,[2] and, as will be pointed out later, a woman with double ovarian tumours may be fertile, for there may be some healthy tissue left.

Class B. Failure of junction of the sperm and the germ cell, and the causes which may be prejudicial.—We do not know where the sperm cell and germ cell meet, though some light has been thrown on the subject in the study of extra-uterine gestation.

1. *In the Male.*

a. **Impotence.** This may occur even in young men, and it is not always the old and decrepit who are affected. It is a most difficult subject of investigation, and its causes are not within the scope of this work.[3]

b. **Deformity**, especially epispadias.

It is important to remember that hypospadias does not cause it.

c. **Gonorrhœa** affecting the male is not considered here, but it acts probably by closing the efferent ducts, and consequently the sperm cell, though produced, cannot escape.

2. *Causes affecting the Woman.*

a. **Vaginismus** (see pp. 33, 34).

b. **Imperforations of the genital tract**, causing impediment to sexual intercourse; these have already been considered.

c. **Imperfect development of the uterus or distortions;**

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[1] Herman, *Diseases of Women*, p. 641.

[2] Champneys, *op. cit.*

[3] Gross on 'Causes of Male Sterility,' *op. cit.*; and Herman, *op. cit.* p. 631.

though mere distortion may mean nothing, as pointed out in the chapter on Minor Uterine Displacements. A woman is not bound to have her uterus exactly in the middle of the pelvis;[1] a uterus may be displaced by old parametric adhesions and yet conception may occur. Anteflexion is the natural shape of the uterus in many virgins,[2] and is no cause of sterility. It is true that an imperfectly developed uterus is often anteverted, but the imperfect development is the cause both of the sterility and the anteversion. Herman[3] has investigated the relations between backward displacements and sterility, and has shown that late in the child-bearing period they are associated with a certain amount of relative sterility, and women having these displacements leave off bearing children sooner, also that their later pregnancies end in abortion oftener than those who have not such displacements.

Herman thinks that it is not a mechanical effect at all, for if so it would be observed in patients of all ages who have these displacements, instead of only the older ones; and he believes the cause is that the state of health which leads to relaxation of the supports of the uterus also leads to exhaustion of the reproductive energy.

The only cases in which retroflexion is a cause of sterility are those in which the body of the uterus is so tender that there is great dyspareunia.

d. **Spasmodic Dysmenorrhœa**[4] is often a sign of poor development of the sexual organs (See chapter on Dysmenorrhœa, where the relations of flexions and versions are also considered.) Flexions are the 'bugbears' of sterility.[5] A woman with acute anteflexion is generally sterile; flexion is a sign of such a uterus not having been impregnated, and of nothing else.[6]

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[1] Matthews Duncan, *Clin. Lect.* 4th ed., on 'Sterility,' p. 170, and on 'Minor Displacements,' p. 407.

[2] Herman, 'Anteflexion and Dysmenorrhœa,' *Trans. Obstet. Soc. Lond.* vol. xxiii. 1881, p. 209.

[3] Herman, *Trans. Obstet. Soc. Lond.* vol. xxxiii. 1891, p. 499; *id. Dis. of Women*, p. 638.

[4] Vedeler, *Arch. f. Gyn.* xix. 1882; Champneys, *op. cit.*, and *Painful Menstruation*, 1891, p. 65; Sir J. Williams, 'The Natural History of Dysmenorrhœa,' *Trans. Obstet. Soc. Lond.* vol. xxiv. 1882, p. 103. (Vedeler's statistics are quoted by Champneys, *Painful Menstruation*, pp. 70–71.)

[5] Matthews Duncan, *op. cit.* p. 170.

[6] Champneys, *Clin. Journ.* Feb. 22, 1893.

Sterility is said to depend upon congenital narrowing of the cervix. No such thing as congenital stricture of the os internum has ever been seen;[1] congenital smallness of the os externum is occasionally seen, so that, instead of being a slit about one quarter of an inch long, it is a little round hole, too small to admit a probe, but this condition is rare.

As Herman says, cases of labour are met with in which great delay takes place in dilatation of the os externum, due, perhaps, to congenital smallness; so that even if it occurs it does not entail sterility.

e. **Occlusion of the genital tract** from inflammatory closure of both tubes (see chapters on Diseases of the Fallopian Tubes, and Pelvic Inflammation).

This is like the occlusion of the vas deferens in man.

f. **Diseased Secretions.**—These may kill the sperm cell, *e.g.* purulent or decomposing material from the cervical canal; gonorrhœa may act in this way, but a woman with gonorrhœa may conceive. Cancer of the cervix is sometimes seen with pregnancy, so that even such discharges may not invariably cause sterility.

Lacerations, erosion, and inflammation of the cervix have been alleged to cause sterility; if this were true large families would be rare. In fact, these conditions are often seen in pregnant women.[2]

Endometritis is often quoted as a cause of sterility, but endometritis is a word 'so loosely used that no one can tell, without description, what anyone means by it' (Herman). Fungous or adenomatous growths are seen in women who have long been sterile, hence their influence is probably adverse to pregnancy.

There is no evidence[3] that hæmorrhage, or catarrhal endometritis, produce sterility, though the former is sometimes associated with abortion, placenta prævia, and accidental hæmorrhage.

Fibroids are said to cause sterility sometimes from the 'endometritis' associated with them, or from the bleeding they give rise to, but pregnancy does occur with fibroids. Probably the only relation of fibroids to sterility is that fibroids occur late in menstrual life, when sterility has already been produced by age. Herman[1] says that fibroids are among the few uterine diseases that affect sterile women and virgins late in life; therefore they seem to affect a large proportion of such women.

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[1] Herman, *Diseases of Women*, p. 633.

[2] Herman, *op. cit.* p. 639.

[3] Herman, *op. cit.* p. 639.

g. **Dyspareunia** may be a cause of sterility, and one of the most frequent ways in which sterility is cured is by the removal of some impediment to complete sexual intercourse.

Dyspareunia means pain or difficulty in coitus, or it may mean both; its chief causes are developmental defects, smallness of the vaginal orifice, vaginismus, and such diseases of the vulva as caruncle, inflammation of the urethra, kraurosis,[2] sore places and fissures of the fourchette or hymen. Vaginitis, and pelvic inflammation accompanied by tender prolapsed ovaries, may also be causes of dyspareunia.

Op. cit. p. 640.

[2] Herman, *Kraurosis Vulvæ*, *op. cit.* p. 621.

CHAPTER X

ON DISEASES OF THE FALLOPIAN TUBES

The Fallopian tubes[1] vary a good deal in length and in different individuals, the average being from three and a half to four inches. The right tube is said to be generally rather longer than the left. The Fallopian tubes run along the upper border of the broad ligaments from the superior angles of the uterus almost to the pelvic brim; they then dip downwards behind the broad ligament and embrace the ovary near its outer end, their direction being first outwards and slightly backwards, then downwards, backwards, and inwards, the fimbriæ finally turning upwards on the outer and posterior surface of the ovary. One of these, the *ovarian fimbria*, is firmly attached to the ovary at its distal extremity. Between the outer end of the Fallopian tubes and the lateral wall of the pelvis is a short fold of peritoneum, the *infundibulo-pelvic ligament*; it is important, as it contains the ovarian artery.

The Fallopian tubes are covered with peritoneum[2] for about five-sixths of their periphery, the remaining and lower one-sixth resting on the connective tissue between the layers of the broad ligaments.

The following portions of the Fallopian tubes are distinguished:

a. The interstitial part piercing the uterine wall.
b. The straight portion, or isthmus.
c. The curved portion, or ampulla.
d. The fimbriated end.

The tube consists of a peritoneal covering a muscular coat in two layers, the inner being circular, and the outer longi-

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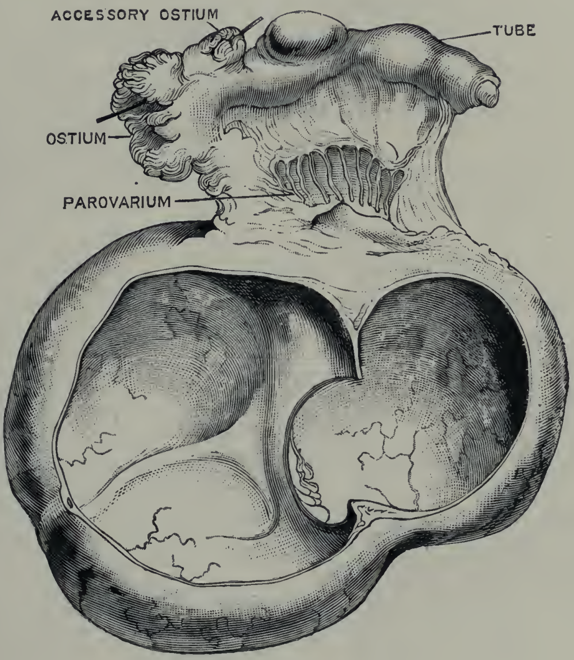
[1] Cullingworth, *Clinical Illustrations of the Diseases of the Fallopian Tubes and of Tubal Gestation*, 2nd ed. preface, p. viii.; Doran, *Encyclop. Med.* vol. iii. p. 429; Cripps, *Ovariotomy and Abd. Surgery*, p. 31.

[2] Berry Hart, Allbutt and Playfair, p. 48.

Fig. 23.

AN OVARIAN CYST SHOWING AN ACCESSORY OSTIUM TO THE TUBE.



An ovarian cyst, with an accessory ostium to the tube. The cyst is a simple multilocular (oöphoritic) glandular adenoma of the ovary, and was the size of an orange. Septa in its interior, showing its original multilocular character, are well shown. The tube on the other side contained an apoplectic ovum, and is shown in fig. 47.

Bland-Sutton, 'Case of Tubal Abortion,' 'Trans. Obstet. Soc. Lond.' vol. xxxii. 1890, p. 343.)

tudinal, and a remarkably folded mucous membrane which connects the genital tract with the peritoneum. The mucosa consists of columnar ciliated epithelium and connective tissue; the foldings are most marked in the ampulla.

Whether these foldings constitute *glands* is still disputed. Berry Hart[1] interprets them as nothing more than a honeycomb arrangement of the tubal lining, indicating, so far as we know at present, its close developmental relation to the uterus. Bland-Sutton,[2] on the other hand, holds that they are glands, and points out that in many mammals the mucous membrane of the tubes is far more complex than in the human female. The grooves and folds of the abdominal ostium can be traced to those within the tube, but secondary plicæ may spring from the sides of the primary, at the ostium.

Accessory Ostia[3] (fig. 23) with fringes and pedunculated tufts of fimbriæ are not uncommon. Bland-Sutton[4] believes them to be displaced Kobelt's tubules. The **hydatid** or **cyst of Morgagni**, derived from the duct of Müller,[5] is a name applied to a small stalked cyst or cysts attached to the fimbriæ or to the tube; such a cyst is rarely larger than a pea. It is not the same thing as the stalked cysts associated with the parovarium or mesosalpinx, derived from Wolffian relics, which have fibrous stalks, and whose walls are lined with cubical epithelium,[6] the true cyst of Morgagni being lined with ciliated columnar epithelial cells.[7]

Atrophy and Hypertrophy of the Tubes.[8]—The tubes atrophy in the ordinary course at the menopause, and they share the processes of involution. Omental adhesions or peritonitic bands may cause atrophy, as may twisting of the pedicle of an ovarian cyst, in which the tube is involved. In large fibroids pressure may also cause atrophy of the tube. Hypertrophy, on the other hand, may occur in pregnancy, or with large fibroids, while stretching of the tube is seen in parovarian tumours, the tube being sometimes of great length. Great thickening may occur in salpingitis.

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[1] Berry Hart, *op. cit.* p. 49.

[2] Bland-Sutton, *Diseases of Ovaries and Fallopian Tubes*, new ed. 1896, p. 197.

[3] Doran, *Trans. Obstet. Soc. Lond.* vol. xxxi. p. 344; also *ibid.* vol. xxviii. p. 171.

[4] Bland-Sutton, *op. cit.*, p. 227.

[5] Bland-Sutton, *op. cit.* new ed. 1896, p. 195.

[6] Ballantyne and Williams, *Brit. Med. Journ.* Jan. 24, 1891.

[7] Bland-Sutton, *op. cit.* p. 229.

[8] Doran, 'Malformations of the Fallopian Tubes,' *Trans. Obstet. Soc. Lond.* vol. xxviii. p. 171, and vol. xxix. p. 186 *id. Encyclop. Med.* vol. iii. p. 430.

Salpingitis, its Causes and Effects.[1]—Inflammation of the Fallopian tubes is nearly always secondary to inflammation of other parts of the genital tract; it is closely allied to endometritis and perimetritis. The chief **causes** are catarrh, septic endometritis occurring mostly after labour or abortion, or other forms of uterine infection, or where the uterus contains septic pathological growths, as cancer or sloughing fibroids. Gonorrhœa[2] in a way seems to be a special source of infection, particularly liable to affect the cervix and tubes. Tubercle also not rarely affects the tubes.

The causes of salpingitis are so intimately bound up with those setting up pelvic peritonitis (perimetritis) that it is unnecessary to separate them, though it is often true that diseased Fallopian tubes are the cause of pelvic peritonitis itself.[3] In the great majority of cases the inflammation starts in the mucous membrane of the uterus, spreading thence to the lining membrane of the Fallopian tubes, and many of the masses formerly described as parametric are now found, on the evidence of the operating theatre and the post-mortem room, to consist of diseased Fallopian tubes[4] surrounded by thickened peritoneum and involved in a mass of adherent viscera, including almost always the ovary and broad ligament, and not infrequently pieces of omentum and one or more coils of intestine;[5] the local peritonitis in such cases being sometimes traced to its focus at the mouth of the tube (Cullingworth).

The Changes in the Tubes.—When inflammation extends from the uterus into the tubes, the tubal tissues become swollen, especially the plicæ, owing to an infiltration of small cells which causes them to become thick and club-shaped. The blood-vessels are at first perhaps dilated, but afterwards appear narrower than in health. Doran[1] points out that there is no rapid desquamation of cells of the plicæ in salpingitis; in fact, this need not take place at all, even in advanced cases, where the tube has been dilated and obstructed for months or years. As the plicæ swell and enlarge by the small cell infiltration, there is adhesion of the edges of adjacent plicæ, but at their roots normal epithelium may remain and spaces are still found lined with healthy epithelium. These spaces are not teratological diverticula, as sometimes described. Deeper still in the middle coat there is œdema and small celled infiltration, this œdema being the chief factor in obstructing the ostium in 'salpingitic closure.'[2] Later the whole tube gets thicker, its normal 'kinks' become more marked, the tube being doubled and folded on itself; the peritoneum also suffers, and there may be local peritonitis around the tube. The inflammation may then run on to the purulent stage, not always forming a pyosalpinx, which implies closure of the tube at its abdominal end; for cases of purulent salpingitis occur without closure, and pus may be squeezed from the open ostium of such a tube.

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[1] Doran, *Encyclop. Med.* vol. iii. p. 431.

[2] Cullingworth, 'Etiological Importance of Gonorrhœa in Diseases of Women,' *Brit. Med. Journ.* July 20, 1889, and *Brit. Med. Journ.* Aug. 12, 1893; Noeggerath, 'Gonorrhœa and Perimetritis,' *Amer. Gyn. Trans.* vol. i. 1876, p. 268.

[3] Cullingworth, 'On the Differential Diagnosis of Pelvic Inflammation in the Female,' *Brit. Med. Journ.* vol. ii. 1890, p. 1468.

[4] Cullingworth, *Clin. Journ.* p. 283, Aug. 30, 1893.

[5] Polk, 'Peri-uterine Inflammation in its Relation to Salpingitis,' *Trans. Assoc. Amer. Physicians Philad.* vol. i. 1886, p. 145.

When this stage is reached, the plicæ are very densely infiltrated and shortened, and may be denuded of epithelium towards the lumen of the tube, but not in the deeper portions and recesses even in advanced cases. Later still, in long-standing cases of pyosalpinx, the plicæ may break down and disappear. At length, the mucosa suffers further damage, and becomes a surface merely covered with granulation tissue like an abscess cavity. Such tubes are not vascular, as the vessels have suffered from the pressure of inflammatory products (Doran).

Sometimes a tube may exhibit all these stages at one time, at one part being fairly healthy, and at another there is advanced inflammation or even a pus-containing loculus. The cyst-like spaces sometimes seen in such tubes, Doran thinks, are to be explained by the persistence of the normal epithelial spaces and crypts at the bases of the inflamed plicæ.

In very long-standing cases of pyosalpinx the pus may degenerate into a watery fluid,[3] the mucosa and muscular coat being merely represented by more or less pure fibrous tissue.

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[1] Doran, Allbutt and Playfair, p. 786. For changes in salpingitis see also Bland-Sutton, *Diseases of Ovaries and Fallopian Tubes*, 2nd ed. 1896, p. 205, and Doran, *Encyclop. Med.* vol. iii. p. 432.

[2] Doran, Allbutt and Playfair, p. 788.

[3] Doran, *op. cit.* p. 791.

Doran says that in chronic catarrhal salpingitis warty growths (papillomata) may develop from the surface of the mucous membrane.[1]

Changes in the Ostium.[2]—Doran has clearly shown that the abdominal ostium of the tube may be closed either by perimetritis or salpingitis.

Perimetritic Closure (fig. 24) is caused by the inflammatory exudation, as in perimetritis, binding down and covering in the delicate fimbriæ; they adhere together and also to surrounding structures; the ostium is thus mechanically sealed up from without. At the same time adhesions bind the tube down to adjacent structures, such as the ovary, broad ligament, uterus, pelvic peritoneum or bowel. This is a protective process against further infection of the general peritoneum by the contents of the tube.

Salpingitic Closure (fig. 25) is closure from within. The fimbriæ are luxuriant protrusions of the mucosa of the tube beyond the ostium; when inflamed they enlarge, the inflammation extends to the muscular coat, which becomes thickened and œdematous, and is lengthened so that it bulges over the fimbriæ until they are more or less covered in. At length the rounded margins of the inner coat meet and cohere, and the fimbriæ can be found folded up inside, roofed over, as it were, by the middle coat.

It is extremely important to remember that the uterine end of the tube is practically never closed, or at all events never in the way above described. It can only be closed by swelling of the mucous membrane, which for a time may be enough to cause obstruction. Following upon closure of the ostium, pus or mucus may accumulate in the tube, which now becomes distended, and the tube assumes the typical retort-shaped swelling so commonly seen with hydro- or pyosalpinx.

Enlargement of the tube is also associated with lengthening and opening up of the mesosalpinx; thus the tube and ovary come into relation with one another, or may even firmly adhere, and such a tube may adhere not only to the ovary, but

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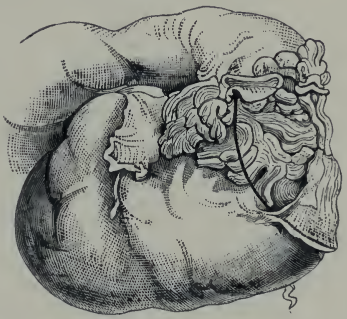
[1] Doran, *Encyclop. Med.* vol. iii. p. 432.

[2] Doran, 'On Closure of the Ostium in Inflammation and allied Diseases of the Fallopian Tubes,' *Trans. Obstet. Soc. Lond.* vol. xxxi. 1889, p. 344.

FIG. 24.

A FALLOPIAN TUBE SHOWING PERIMETRIC CLOSURE OF THE OSTIUM.

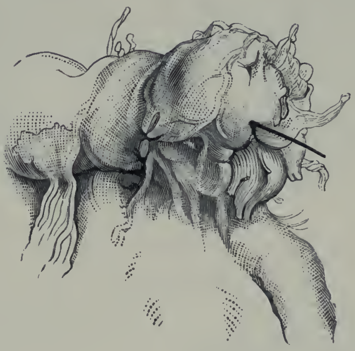


An ovary and tube showing obstruction of the ostium by perimetric deposit, which forms a deep pouch around the orifice of the tube. The fimbriæ have been partly pulled out of the pouch. A bristle passes into the pouch out of the ostium.

(Doran, 'On Closure of the Ostium in Inflammation, and Allied Diseases of the Fallopian Tube,' 'Trans. Obstet. Soc. Lond.' vol. xxxi. 1889, p. 358.)

Fig. 25.

A FALLOPIAN TUBE SHOWING SALPINGITIC CLOSURE OF THE OSTIUM.

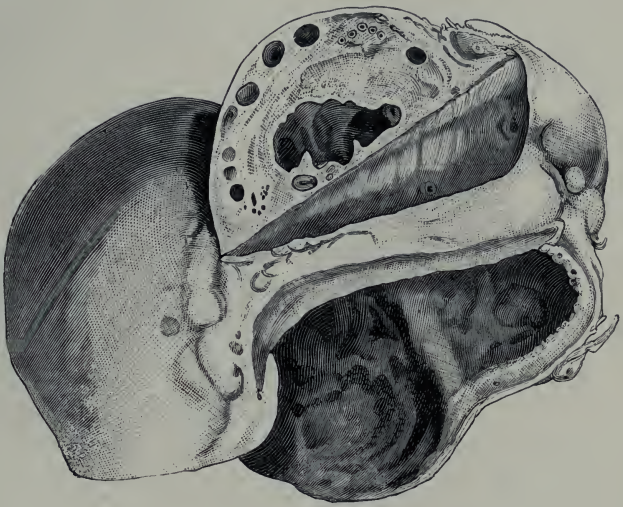


A specimen showing complete obstruction of the ostium, the result of salpingitis. The end of the tube has been detached from the ovary below, and the ostium forcibly opened; a bristle passes out of its orifice. The tissues of the tube have swollen over the ostium, completely concealing the fimbriæ excepting the ovarian fimbria, which is seen below the bristle. Behind and above the bristle are perimetric bands, which must not be mistaken for fimbriæ.

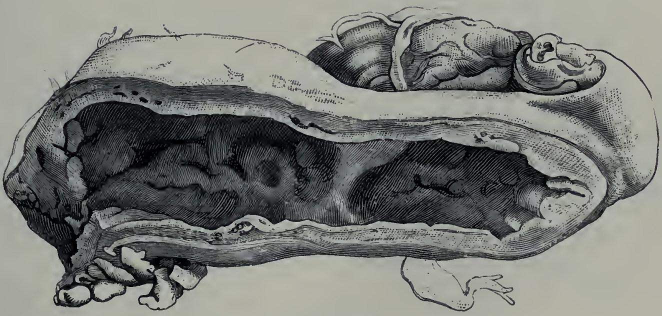
(Doran, 'Trans. Obstet. Soc. Lond.' vol. xxxi. 1889, p. 358.)

FIG. 26.

PURULENT SALPINGITIS DUE TO GONORRHŒA.



Right Tube.



Left Tube.

Two Fallopian tubes removed by operation from a woman of 25. Both tubes are enormously enlarged and irregularly dilated; the right measured 4½ inches, and the left 6½ inches in circumference; both have greatly thickened walls, with ulceration of the mucous lining, and were filled with thick yellow pus. The abdominal ostium of each is occluded.

(Specimen 2391. Museum, St. Thomas's Hospital. Drawing by W. E. Roth. Cullingworth, 'Trans. Obstet. Soc. Lond.' vol. xxxiv. 1892, p. 373, and 'Diseases of Fallopian Tubes,' plate vii.)

also to any of the adjacent structures. By its weight, it tends to fall down behind the uterus, and it is not uncommon to find, in chronic cases of pelvic peritonitis, masses firmly bound down behind the uterus consisting of such tubes and ovaries adherent to everything—broad ligament, uterus, floor of the pelvis, or bowel. At operations such tubes may be very difficult to recognise and still more difficult to remove. Among the adhesions curious collections of serum occur; they are intraperitoneal.[1]

Pyosalpinx is the name given to such tubes when distended with pus. On section [2] they are seen often to consist of a series of loculi or pouches; generally these are in communication with one another, the intervening portions of the tube remaining pervious (fig. 26); but occasionally, owing to the sudden bends in the tube, or from constrictions by peritoneal bands or adhesions within the tube, the pockets get shut off, forming independent collections of pus.

Sometimes the ovary in cases of salpingitis is normal, except for its adherence to the tube; in others the ovary is in a condition of incipient cystic degeneration; sometimes such cysts are as large as a marble, and the whole ovary is enlarged and can be grasped. If such cysts become infected from the tube the ovary suppurates and a well-marked **abscess** forms in it. This is not infrequently seen in gonorrhœal cases.[3] Sometimes the pus-containing tube becomes adherent to a cystic ovary, the wall of the tube ulcerates, perforation occurs, and pus escapes into the interior of the cyst and a **Tubo-ovarian abscess** results.[4] Sometimes a pyosalpinx very much resembles this condition; there is a sudden retort-like expansion of the end of the tube, and, unless care be taken, it may be thought to be ovarian tissue, while further search may discover the ovary to be quite healthy. Cullingworth refers to such a specimen in St. Thomas's Hospital Museum (No. 2396).

Pyosalpinx may sometimes attain a very large size and form huge masses in the abdomen as high as the navel, containing many ounces of pus.[1] These very large specimens are certainly rare, and are probably tubercular ;[2] they do not seem to be of septic origin ; they are banana-shaped[3] instead of the usual retort-shaped, sacculated tumours, common in ordinary pyosalpinx. These banana-shaped tubes resemble pyometra or hydrometra in bicornute uteri,[4] but the evidence of inflammation in the former settles their origin. What eventually happens in cases of pyosalpinx is a little uncertain. Rupture may occur[5] in a few rare cases, and fatal peritonitis ensue, but its frequency is exaggerated.[6] The tube may also ulcerate from within, and the pus escape into a space bounded by adhesions (which is certainly commoner), generally in Douglas's pouch. In pregnancy, when the tubes are high up, such an abscess may be above the brim. Sometimes in pyosalpinx, the pus finds its way into adjacent bowel, usually the rectum. Many intraperitoneal abscesses[7] start in connection with inflamed suppurating tubes of this nature.

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[1] Targett, 'Perimetric Cystoma,' *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 343.

[2] Cullingworth, *Clinical Illustrations of the Diseases of the Fallopian Tubes and of Tubal Gestation*, 1896, p. 67.

[3] Cullingworth, 'Pelvic Peritonitis attended with Abscess of the Ovary,' *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 277.

[4] Cullingworth, *Clin. Journ.* Aug. 30, 1893, p. 282.

Pyosalpinx not infrequently accompanies cancer of the uterus,[8] and may cause death in such cases.[9] Bland-Sutton says that pyo- and hydrosalpinx complicate uterine cancer in the proportion of not less than ten per cent.[10]

Chronic Salpingitis.—Whether or not such cases can be absolutely separated from chronic pelvic peritonitis is uncertain, and in many cases it is impossible to do so. There are a large number of women coming to the out-patient rooms of every large hospital with chronic pelvic pain and inflammatory trouble dating often from some previous confinement, and on examination some thickening may be found in Douglas's pouch

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[1] Roberts, *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 121. Specimen, St. Bartholomew's Hospital Museum, 2936 B.

[2] Targett, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 341.

[3] Bland-Sutton, *Diseases of Ovaries and Fallopian Tubes*, 2nd ed. 1896, p. 214.

[4] Bland-Sutton, *op. cit.* 1st ed. p. 248.

[5] Lewers, *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 298.

[6] Champneys, 'On Removal of Uterine Appendages,' *St. Bartholomew's Hosp. Reports*, 1893, p. 53, p. 61, and in Discussion ; Cullingworth, 'On the Value of Abdominal Section in certain Cases of Pelvic Peritonitis,' *Trans. Obstet. Soc. Lond.* vol. xxxiv. 1892, p. 430.

[7] Cullingworth, 'Pelvic Abscess,' *Birmingham Med. Review*, Nov. 1893 ; *id.* Allbutt and Playfair, 'Pelvic Inflammation,' p. 505 ; *id. Brit. Med. Journ.* vol. ii. 1893, pp. 353 and 981.

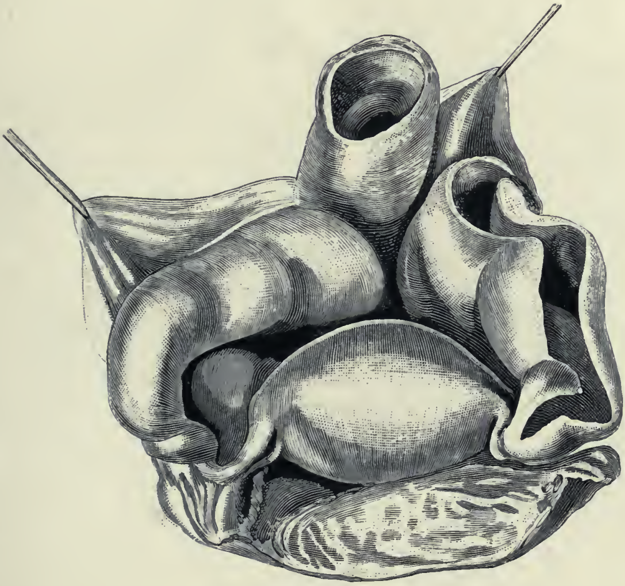
[8] Lewers, *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 199.

[9] J. K. Fowler, 'A Contribution to the Pathology of Hydro- and Pyosalpinx,' *Proc. Med. Soc.* vol. vii. 1884, p. 441.

[10] Bland-Sutton, *op. cit.* 2nd ed. 1896, p. 213.

Fig. 27.

DOUBLE HYDROSALPINX.

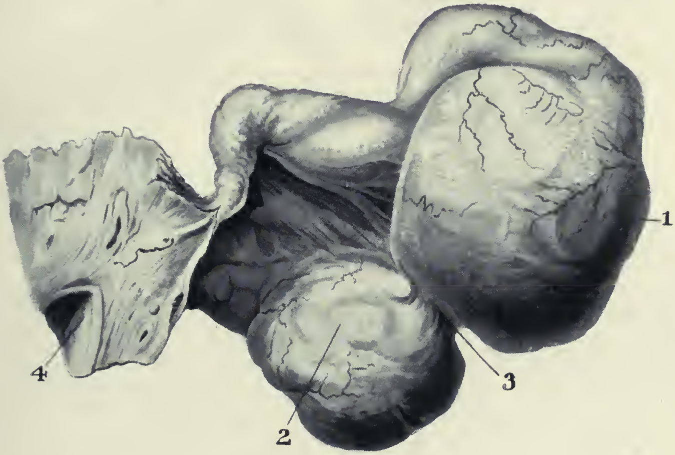


Specimen showing great distension of the Fallopian tubes. The abdominal orifices are closed. The extremities of the distended tubes lie in Douglas's pouch, where there are firm adhesions. Sections of the uterine end of the tubes showed the mucous membrane to be partially destroyed.

(Griffith, 'Trans Obstet. Soc. Lond.' vol. xxxiii. 1891, p. 158.)

FIG. 28.

HYDROSALPINX AND CYSTIC OVARY.



1. Fallopian tube which was distended with clear fluid to form a hydrosalpinx. It shows the typical retort-shape which such tubes generally assume.

2. The ovary, which is also cystic.

3. An adhesion between the ovary and the distended tube, showing the possible formation of a tubo-ovarian cyst should the ovary and tube communicate.
4. Portion of uterus, showing its cavity. The uterus contained several fibroids.

(Specimen 2963 A. Museum, St. Bartholomew's Hospital.)

as evidence of previous inflammation. Such cases are better from time to time; some recur; some, in spite of the pelvic condition, have no symptoms; some, on the other hand, get worse, their life becomes a burden from constant pain, and they are unable to follow the ordinary vocations of life. Many of these have never been properly treated, and a rest in hospital may cure them, or at least they are so much improved that they do not trouble further in the matter.

Many of such cases doubtless depend on chronic inflammation of the tubes; some are evident, some are not, and the disease is perimetritis; but perimetritis does not kill in itself, though the disease causing the perimetritis may do so, such as ovarian tumours or fibroids. Even in tubal affections as a cause, the mortality is very low, and, as previously stated, the dangers of general peritonitis from bursting of a tube are greatly exaggerated.[1] It would be perhaps out of place in a work on pathology to discuss treatment, but great harm has resulted from too frequent operation in cases of chronic salpingitis (or perimetritis). Certainly early operation, before proper medical treatment has been tried, is unjustifiable; there are risks of operation, and the results of removal of the appendages are certainly grave and not to be lightly undertaken; and again, useful as such an operation may be in salpingitis, it is only suitable for some chronic cases and rarely for any others. Further, the mortality of the class of case for which it is recommended is, apart from operation, very small indeed.

Hydrosalpinx. (Fig. 27.)—This is distension of the tube by watery fluid. It may be a late stage of pyosalpinx, as Bland-Sutton states,[2] but it is quite possible that not all cases of inflamed occluded tubes run on to the formation of pus, but that catarrh of a mild type may seal the ostium and then the secretions of the tube itself cause distension. They are in fact retention cysts, the tube being stretched and thinned, and the mucosa atrophied till nothing but a thin-walled transparent cyst remains.[3] A hydrosalpinx is generally more or less retort-shaped (fig. 28) and curves round the ovary; sometimes a dimple (fig. 29) exists where the ostium was, from which furrows and folds radiate. Cullingworth [1] explains the closure of the ostium, not so much from an inflammation of the tube itself, but rather that it results from a peritonitis originating in disease of some other part of the pelvis,[2] not infrequently in purulent inflammation of the tube of the opposite side. Hydrosalpinx may attain great size, but it is not common: small examples are seen not infrequently with uterine fibroids. The fluid of a hydrosalpinx is generally clear serum, or it may be coloured, and is generally harmless if rupture should occur. Hydrosalpinx may, like an ovarian cyst, undergo axial rotation.

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[1] Champneys, 'On the Removal of the Uterine Appendages,' *St. Bartholomew's Hosp. Reports*, vol. xxix. 1893, p. 61.

[2] Bland-Sutton, *Diseases of Ovaries and Fallopian Tubes*, 2nd ed. 1896, pp. 216–220.

[3] Bland-Sutton, *ibid.* p. 218.

Hydrops Tubæ Profluens is considered with Papilloma of the Tube.

Hæmatosalpinx.—This is a term applied to Fallopian tubes closed at the abdominal ostium and distended with blood. It must be admitted that a large number of such cases are connected with tubal pregnancy, and much discussion has arisen on this point. Probably it would be wiser to include under this term all effusions of blood into the Fallopian tube, from whatever source.[3] Not all tubes containing blood are necessarily closed [4]; if open, they are practically all due to early tubal pregnancy (see figs. 42, 49), the tube generally containing a mole (see figs. 44, 45). Bland-Sutton says that nearly all specimens supposed to be examples of hæmatosalpinx are really gravid tubes.[5] Possibly in the majority of cases this is true and the evidence is complete; but there are other cases [6] in which the evidence on the clinical side is strong, but no distinctive products of conception are found (figs. 43, 48, 49) to make the case conclusive at operation. Again, in another group of cases the presumptive evidence may be inconclusive on either side; and lastly, the evidence may point to other causes than tubal gestation, as in cases where blood is found in the tubes in some cases of pelvic inflammation, or in cancer of the tube or uterus, or where fluid blood is found only in one

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[1] Cullingworth, *Clinical Illustrations of the Diseases of the Fallopian Tubes and of Tubal Gestation*, p. 1, plate 1.

[2] See also Doran, *Encyclop. Med.* vol. iii. p. 433.

[3] Cullingworth, 'Effusions of Blood into the Fallopian Tubes,' *St. Thomas's Hosp. Reports*, vol. xxi. p. 23.

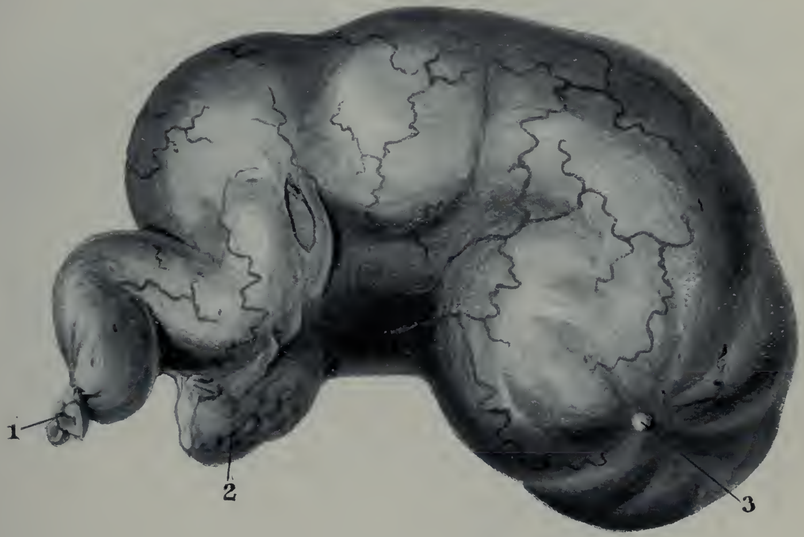
[4] Doran, *Encyclop. Med.* vol. iii. p. 434.

[5] Bland-Sutton, *op. cit.* (1891), p. 262.

[6] Cullingworth, *St. Thomas's Hosp. Reports*, vol. xxi. p. 23.

Fig. 29.

HYDROSALPINX.



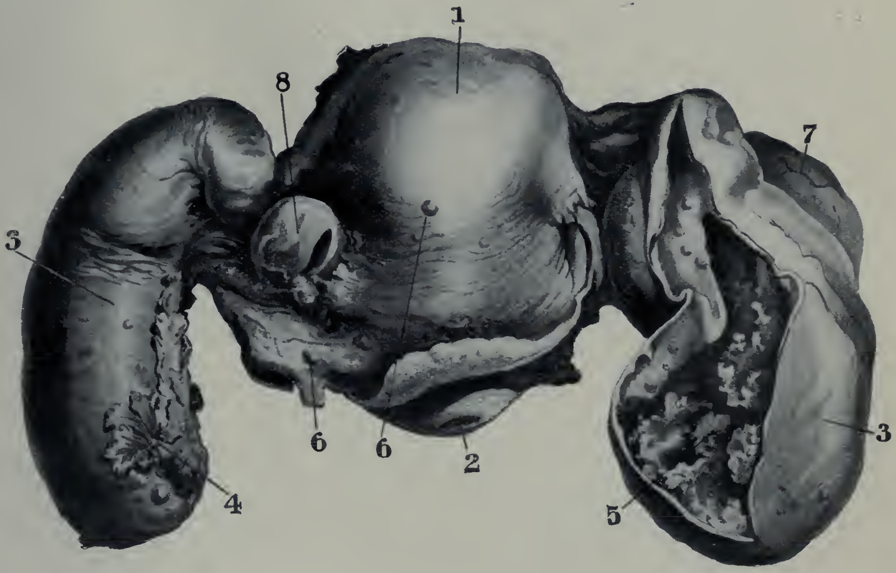
A much-distended and convoluted Fallopian tube removed from a woman of 49, who had suffered with pelvic peritonitis. It exhibits the usual retort-shaped expansion of the tube, and contained clear fluid.

1. Uterine end of the tube.
2. Ovary.
3. Fimbriated extremity of the tube represented by a dimple; all traces of the fimbriæ are lost.

Specimen 2382 a. Museum, St. Thomas's Hospital. See also Cullingworth, 'Clinical Illustrations of Diseases of the Fallopian Tubes,' plate i. fig. 1.)

Fig. 30.

TUBERCULAR DISEASE OF THE FALLOPIAN TUBES.



A uterus and Fallopian tubes; the latter are enlarged, and form two banana-shaped swellings which do not resemble the usual retort-shaped tumours usually seen in pyosalpinx. The tubes are closed at their fimbriated extremities. The right tube has been laid open; it is occupied by cheesy deposits, and in places is ulcerated and thickened. It contained pus. Here and there definite tubercles are seen on the peritoneal surface of the uterus and tubes.

1. Fundus uteri.
2. Cervix.

3, 3. The distended tubes. Note the banana-like shape.

4. Fimbriated extremity of the left tube.
5. Right tube opened to show cheesy deposit in its interior.

6, 6. Tubercles beneath the peritoneum.

7. Enlarged ovary on the right side.
8. Mass of breaking-down tubercular material.

Transverse sections of the undilated uterine end of the tubes showed abundant infiltration of the mucous membrane with grey and caseous tubercle possessing well-defined giant-cell systems.

(Targett, 'Trans. Obstet. Soc. Lond.' vol. xli. 1899, p. 165, who kindly allowed the author to draw the specimen.)

tube, and a tubal gestation in the other. Hæmatosalpinx associated with hæmatometra is a grave condition.

Tuberculosis of the Fallopian Tubes.[1]—The tubes are by far the most frequent seat of genital tuberculosis in the female, and the majority of cases are secondary to tuberculosis elsewhere, either by infection from neighbouring organs, or from the blood; and even in primary cases it is impossible to exclude blood infection. Whether genital tuberculosis can result from vaginal infection by digital examination, instruments, or coitus, has not yet been satisfactorily proved.

Tubercular salpingitis may be either in the form of acute miliary, or chronic diffuse tuberculosis;[2] fibroid tuberculosis is very rare.

Microscopically, in its early stages tubercular salpingitis resembles the ordinary inflammatory form;[3] in fact, inflammation may precede it, and the epithelium, though swollen, is late in being shed, the essential change being diffuse cell-growth of a lymphoid and epithelioid character in the plicæ. Next follows cheesy metamorphosis of this cell-growth, beginning in the nuclei of the epithelioid cells. Later, when caseation is well marked, the epithelium of the plicæ is destroyed secondarily and the diffuse cell-growth spreads to the muscular tissue of the wall of the tube, with thickening and subsequent breaking down of the tissues, exhibiting the characteristic elements of tubercular disease.[4]

Doran finds that even in late cases the same cyst-like areas are found, representing spaces between the roots of the plicæ, as described in ordinary salpingitis. At length, the whole tube becomes broken down, with marked caseation and deep tubercular ulceration, and pus forms either in loculi, or distends the whole tube. Sometimes such a tube is not very large, being long and tortuous, but in other rare cases large banana-like or legume-shaped tumours (fig. 30) are formed, not at all like ordinary pyosalpinx in shape, with the interior almost smooth, and unless careful sections are made of the uterine end of such tubes their tubercular origin may not be discovered.[1]

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[1] Whitridge Williams, 'Tuberculosis of the Female Generative Organs,' *Johns Hopkins Hosp. Reports*, vol. iii. 1892; also P. Kidd, 'Lesions of Phthisis,' *Med. Chir. Trans.* vol. lxviii. 1885, p. 87, and *Trans. Path. Soc.* vol. xxxvii. 1886, p. 357; Turner, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 161.

[2] Bland-Sutton, *op. cit.* 2nd ed. 1896, p. 209.

[3] Doran, Allbutt and Playfair, p. 796.

[4] Doran, Allbutt and Playfair, p. 795.

In many cases of tubercular disease of the tubes, whether primary or secondary, the pelvic peritoneum is affected, sometimes to a very great extent. There is enormous thickening, matting, and distortion of all its folds, especially the anterior pouch, so that even the viscera are difficult to recognise, being bound down amidst adhesions. In some of these cases it is much more probable that the peritoneum is affected primarily,[2] for the tubes, though bound down, may be found free from disease, and not even closed at their fimbriated end.

Tubercular salpingitis is a very chronic disease, and ultimately the result may be the same as in neglected pyosalpinx from other causes, and the pus may find its way out by opening into viscera, leaving discharging fistulæ; or general systemic tuberculosis may ensue. Ascites is very frequent in tubercular peritonitis. Tubercular disease of the Fallopian tubes may be found in very young children. Silcock's[3] case occurred in a child of five, and Percy Kidd's[4] in a girl of fourteen.

Hydatids of the Fallopian Tubes and Actinomycosis.—These must be very rare conditions, but one or two have been described. Probably in actinomycosis[5] the peritoneal infection is the primary factor.

Fibroma and Enchondroma.—It is very doubtful if either such tumours have been detected. Sometimes the fimbriæ contain gritty particles which Doran thinks identical with similar bodies found in papillomatous cysts of the ovary.

Myoma of the Fallopian Tubes.[6]—Authentic cases of myoma of the tube are rare,[7] but it is possible that such tumours might occur, as the tube is morphologically part of the uterus, and, as most reported cases are of the pedunculated variety, it is possible they might have developed from the broad ligament

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[1] Targett, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 163.

[2] Targett, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 116.

[3] Quarry Silcock, *Trans. Path. Soc.* vol. xxxvi. 1885, p. 303.

[4] Percy Kidd, *op. cit.*

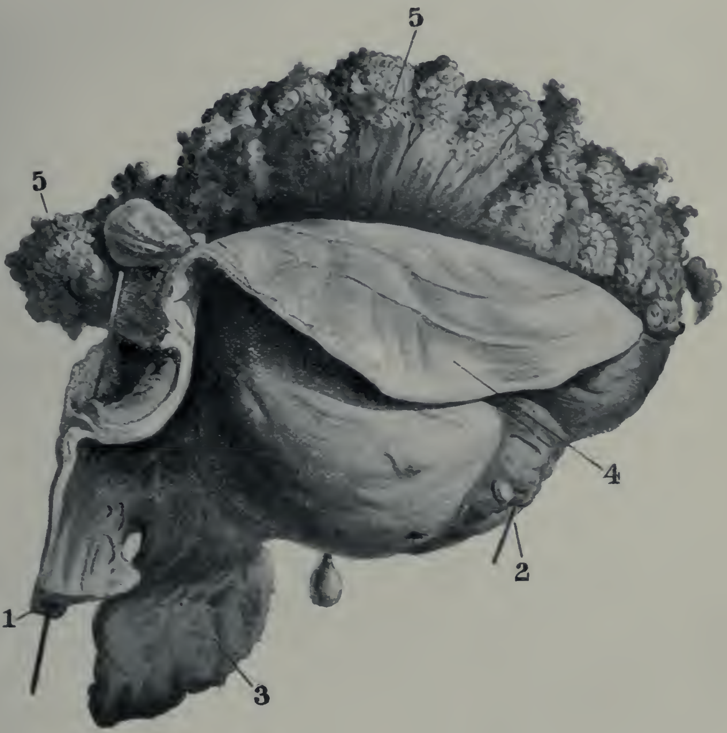
[5] Doran, *Encyclop. Med.* vol. iii. p. 431; Zemann's case, quoted by Doran, Allbutt and Playfair, p. 798; *Med. Jahrbücher*, Wien, 1883, p. 477; Doléris, 'Echinococcus of Fallopian Tube,' *La Gynécologie*, April 15, 1896.

[6] Doran, Allbutt and Playfair, p. 799.

[7] Simpson, *Clin. Lect. on Dis. of Women*, 1872.

FIG. 31.

PAPILLOMA OF THE FALLOPIAN TUBE.



A Fallopian tube enlarged and dilated by masses of papillomatous growths, which spring from the mucous membrane. They have not rendered the tube impervious, and a bristle is passed completely through it from the uterine end to the fimbriated extremity. During life a mucoid discharge issued from the tube and set up ascitic and pleural effusion. No secondary deposits were found, and the patient is alive now, twenty-two years after the operation.

1. Uterine end of tube. A bristle is passed through this to the fimbriated extremity.
2. Bristle issuing from fimbriated extremity of tube.
3. Ovary, which is healthy. A small pedunculated cyst is seen below the tube near the ovary.
4. Wall of tube, turned down to show growth inside.

5, 5. Masses of papillomatous growth filling the tube.

(Specimen 4584. Museum, Royal College of Surgeons. Doran, 'Trans. Path. Soc. Lond.' vol. xxxi. 1880, p. 174, and Allbutt and Playfair's 'System of Gynæcology,' p. 806, and No. 1, Tables, p. 829. Compare No. 5 in the same tables, where there was hydrops tubæ profluens [Doléris].)

(Doran). Bland-Sutton emphasises Virchow's statement that myomata originating in the muscular tissue of the Fallopian tube are very rare.[1]

Cysts of the Fallopian Tubes.—Small cysts of the fimbriæ are not at all uncommon, the largest being known as the Hydatid of Morgagni, which is without doubt a tubal appendage, and is lined with endothelium. Doran[2] figures a remarkable case of Sänger's, where two masses of cysts and solid myxomatous growths, probably of congenital origin, sprang each from a pedicle which was an abnormal fimbria.

Dermoid Tumours of the tube are without doubt exceedingly rare.

Fatty Tumours in the region of the tube probably arise in the broad ligament, and afterwards invade the tube.

Papilloma and Adenoma of the Tube.[3]—Doran first applied the term papilloma to an exuberant morbid growth in the interior of a Fallopian tube; while Bland-Sutton[4] classes the same growth as an adenoma, on the supposition that 'if the mucosa of the tube be glandular, the occurrence of adenoma is not only possible, but probable.' Doran says that such growths commence in the mucosa as villi or papillæ,[5] and that these elevations are not glands or tubal folds. Later, there is great increase in the stroma, which makes the tumour assume the appearance of a succulent adenoma, and eventually the whole tube may be filled with a sprouting mass of soft growth, consisting of papillæ covered by ciliated columnar epithelium growing from all parts of the mucosa, but not invading the walls. (Fig. 31.) The tube may also contain much watery mucoid fluid secreted by the growth.

The occurrence of profuse watery secretions in papilloma of the tube is to be noted. The ostium may be very patulous, and there is sometimes hydroperitoneum, probably from irritation of the peritoneum itself, or, according to some authorities, from the watery discharges escaping from the tube through the patent ostium into the peritoneal cavity.

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[1] Bland-Sutton, *Tumours, Innocent and Malignant*, p. 142; *id. Diseases of Ovaries and Fallopian Tubes*, 1896, p. 228, with reference to Spaeth's case.

[2] Doran, Allbutt and Playfair, p. 802; Sänger and Barth, Martin, *Die Krankheiten der Eileiter*, Berlin, 1895.

[3] Doran, *Trans. Path. Soc.* vol. xxxi. p. 174; *ibid.* vol. xxxviii. p. 241; *ibid.* vol. xxxix. p. 201; *Trans. Obstet. Soc. Lond.* vol. xxviii. p. 229.

[4] Bland-Sutton, *Diseases of Ovaries and Fallopian Tubes*, 1891, p. 280; *id.* 'Glands of the Fallopian Tubes,' *Trans. Obstet. Soc. Lond.* vol. xxxii. 1890, p. 189.

[5] Eberth and Kaltenbach, *Zeitschr. f. Geburts.* xvi. 1889, p. 357.

It is said that if the ostium be blocked these watery discharges (**Hydrops Tubæ Profluens**)[1] occur from the uterus and vagina, in gushes, the uterine end of the tube from time to time allowing the passage of the fluid, but at present there is no trustworthy proof that the fluid necessarily comes from the tube.[2]

It appears that not infrequently a history of previous inflammation of the Fallopian tube is attainable in papilloma; and it is possible that, in tubes previously diseased, papilloma may develop as the result of salpingitis.

The question of the innocency or malignancy of these growths is of great importance. The disease is known to assume characters apparently malignant, and yet the after history, on removal of the growth, may prove to be innocent; 'it seems equally certain that if left alone the papilloma will undergo malignant degeneration' (Doran).

Cancer of the Fallopian Tubes. (Figs. 32, 33, 34.)—There is no doubt that Primary Cancer of the Fallopian tube[3] occurs, while, rarely, extension of cancer may take place to the tubes from the uterus or ovaries.

Doran, whose admirable work on the subject should be consulted thinks that, malignant disease of the tube may be the result of degeneration of papilloma of the tubal mucous membrane.

Bland-Sutton,[4] upholding the other view, states that, as the tubes may be the seat of adenoma, it leads us to expect that they might occasionally be affected with primary cancer. Over thirty cases have been reported.[5]

Probably in primary cancer of the Fallopian tubes two forms may be recognised.[6]

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[1] Doran, Allbutt and Playfair, p. 806.

[2] Bland-Sutton, *Diseases of Ovaries and Fallopian Tubes*, 1891, p. 259; *ibid.* 2nd ed. 1896, p. 227; *Intermitting Hydrosalpinx*, p. 221.

[3] Doran, 'Primary Cancer of the Fallopian Tube,' *Trans. Path. Soc. Lond.* vols. xxxix., xl., and xli.; also *id.* Allbutt and Playfair, *System of Gynæcology*, p. 812, with tables and references; and *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 137.

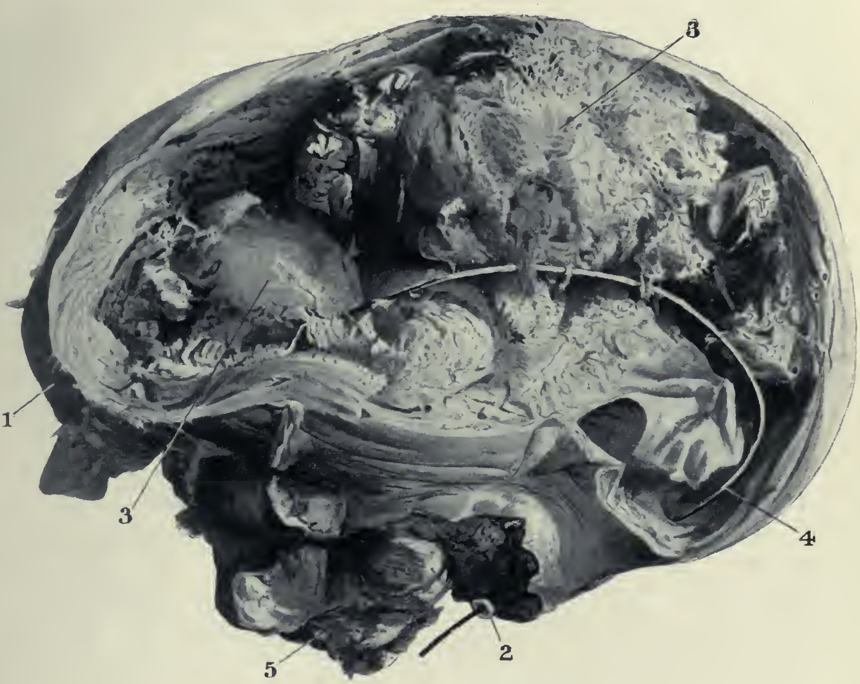
[4] Bland-Sutton, *Surgical Diseases of Ovaries and Fallopian Tubes*, 1896, p. 230; Sänger and Barth, in Martin's *Krankheiten der Eileiter*, Berlin, 1895, quoted by Bland-Sutton.

[5] Boursier and Venot, *Revue de Gynéc. et de Chir. Abdom.* vol. v. 1901, p. 223.

[6] Doran, Allbutt and Playfair, p. 812.

Fig. 32.

PRIMARY CANCER OF THE FALLOPIAN TUBE.



A much-enlarged Fallopian tube, showing masses of cancerous growth springing from the mucous membrane; the growth is flocculent on its free surface, and at certain points has invaded the muscular wall of the tube. The ovary is converted into a mass of tumour substance, and the broad ligament near the uterine end of the tube is also infiltrated with new growth.

1. Fimbriated extremity of the tube; the wall is thickened and infiltrated with growth.
2. Uterine end of tube; a bristle (4) is seen passing through it into the lumen of the tube.

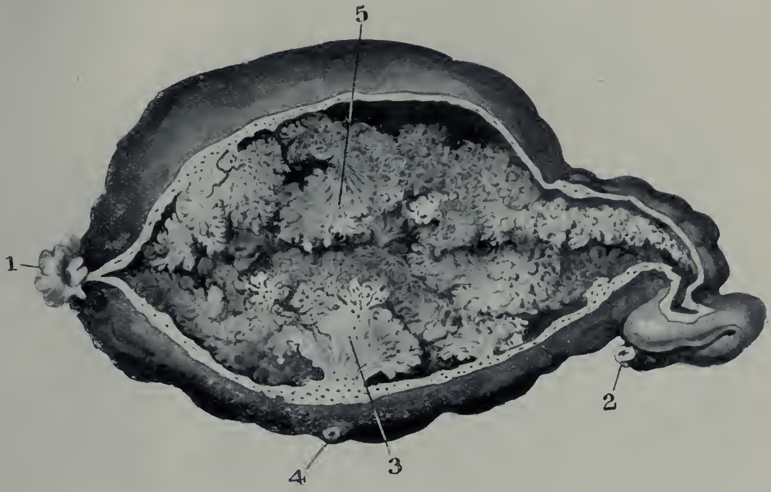
3, 3. Masses of cancerous growth, which under the microscope showed large polymorphous cells bounded by trabeculæ, the growth having the type of a papillary carcinoma. The patient died of recurrence eleven months after the operation for its removal.

5. Remains of ovary infiltrated with growth.

(Specimen 4584 d. Museum, Royal College of Surgeons. Doran, 'Allbutt and Playfair's System of Gynæcology,' article 'Diseases of Fallopian Tubes,' p. 814, Case 2 in Cancer Series, p. 830; *id.* 'Trans. Path. Soc. Lond.' vol. xxxix. 1888, p. 208, and vol. xl. 1889, p. 221.)

Fig. 33.

PRIMARY CANCER OF THE FALLOPIAN TUBE.



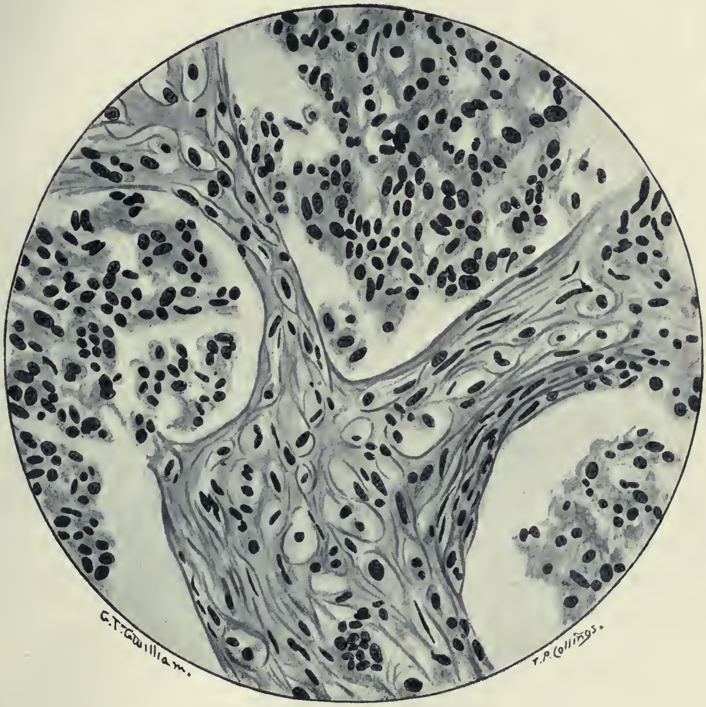
A Fallopian tube much enlarged and distended by a mass of cancer. The whole tube is filled with growth to within an inch of the uterine end; in places the walls are invaded to the peritoneal surface. The lumen of the tube was filled with sanious fluid. The age of the patient was 43; recurrence took place eight months after the operation. The tube was the size of a Bologna sausage.

1. Fimbriated extremity of the tube, which is closed.
2. Uterine end of tube.
3. Mass of cancer invading the wall of the tube; a nodule (4) has reached the peritoneal surface.
5. Masses of flocculent breaking-down growth occupying the lumen of the tube.

(Specimen 2938 G. Museum, St. Bartholomew's Hospital. Roberts, 'Trans. Obstet. Soc. Lond.' vol. xl. 1898, p. 189; *id.* 'Trans. Obstet. Soc. Lond.' vol. xli. 1899, p. 129.)

Fig. 34.

PRIMARY CARCINOMA OF THE FALLOPIAN TUBE.



Section of the deeper portions of the growth in the tube-wall, from specimen shown in fig. 33. It shows clusters of carcinomatous cells gathered in irregular lacunæ; some have involved the connective tissue of the tube beneath, but the growth was strictly limited to the tube itself.

(Specimen 2938 G. Museum, St. Bartholomew's Hospital. Roberts, 'Trans. Obstet. Soc. Lond.' vol. xl. p. 194; see also Doran's series, *ibid.* Case 20, p. 204.)

a. Carcinoma developing in the mucosa of a normally formed tube.

b. Developing in a tube which is malformed, bearing a cyst (not connected with the ovary) into which the ostium opens.

A factor of extreme importance is, that, in many cases of cancer of the tubes, there appears to be a distinct history of previous chronic inflammation. This has also been noted in connection with papilloma; at all events, morbid papillæ are prone to develop in chronically inflamed tubes, and from such morbid papillæ cancer may arise. In cancer of the tube, as in papilloma, the condition called Hydrops tubæ profluens has been observed.

Primary Cancer developing in a Normal Tube.—*a.* These cases resemble one another more or less. The tube is generally somewhat enlarged and thickened, and in advanced cases bound down to surrounding structures, possibly previous inflammation playing some part; often the shape of the tube is altered, no trace of the ostium can be discovered, and the ovary may be infected with the same growth by extension. The tube is dilated and filled everywhere with delicate papillary masses (fig. 32) which are soft and spongy, very vascular, and bathed in thinnish, watery, mucoid fluid. The portions of the growth towards the lumen of the tube are often very necrotic and macerated; here and there distinct tufts of growth can be seen springing from a thickened, infiltrated portion of the tube wall (fig. 33); in other parts the wall may be thin and sacculated.

Under the microscope, these projections have the character of villi with delicate connective tissue stems, covered with cylindrical epithelium, which towards the lumen of the tube are very œdematous and degenerate. Deeper, the epithelium is seen invading the stroma of the villous processes, and passing in groups and columns, deep into the mucosa and muscle, and even reaching the peritoneal aspect. In many cases the columnar type of the cells is lost, and they assume a polymorphous type, lying in alveoli (fig. 34). Sometimes, in the deeper parts, Doran noted tubules lined with perfect columnar epithelium, and surrounded by a wide area of cancer cells; their exact explanation seems impossible. Doran[1] thinks they are similar to the cysts lined with epithelium described in salpingitis resulting from the inclusion of remnants of normal epithelium at the base of the plicæ.

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[1] Doran, Allbutt and Playfair, p. 814.

Cancer of the Fallopian tube is sometimes seen with an ovarian cyst.[1]

b. **Primary Cancer developing in a Cyst connected with the Ostium.**—This is very rare, and Doran says only two cases have been noted. Its pathology is obscure. In Wynter's case the tube was malformed, its ostium opening into a distinct cyst, which, unconnected with the ovary, was probably an abnormal (congenital ?) condition.[2]

Clinically, cancer of the tube, at least in its early history, suggests tubal inflammation. It never occurs in youth; out of Doran's seventeen cases, only one was as young as thirty-six, another forty-three, all the others over forty-five. In two cases reported by the author[3] since Doran's first seventeen cases, the ages of the patients were forty-eight and sixty. Recurrence took place in both cases, in one within eight months, and in the other within three years; in these cases the peritoneal cavity was the seat of the recurrence. The type of malignancy, however, in cancer of the tubes does not appear to be very high, and for a long time the growth seems to be confined within the limits of the tube. Pawlik, of Prague, recently reported (*Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 6) a case occurring in a multipara aged seventy suffering also from endometritis and colpitis granulosa. The tube and ovary (right) were removed with the uterus; the patient died of recurrence two years and five months later.

Doran[4] remarks with regard to age, that when a patient who has reached her forty-fifth year, and who has been subject to pelvic inflammation, shows a sudden or steady aggravation of subjective and objective symptoms, cancer of the tube may be suspected; a watery or especially a sanious discharge in such a case greatly increases the probability of malignancy.

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[1] Cullingworth, *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 307, plate 5, p. 310.

[2] Essex Wynter, *Trans. Path. Soc.* vol. xlii. 1891, p. 221. Doran is now of opinion that the authors of the reports of these cases may have taken the outer part of a hydrosalpinx or pyosalpinx for a cyst distinct from the tube (see above, p. 137).

[3] Roberts, 'Primary Carcinoma of the Fallopian Tube,' *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 189, and vol. xli. 1899, p. 129.

[4] Doran, Allbutt and Playfair, p. 823.

Sarcoma of the tube[1] as a primary growth is very rare, though cases have been reported of this nature,[2] but, as Doran says, the difficulty of distinguishing between new growths and inflammatory deposit greatly complicates the sarcoma question on account of the well-established frequency of inflammatory changes preceding the development of a true neoplasm. Nothing can be decided until there is more clinical evidence at our disposal, and much the same must be said of Deciduoma malignum of the tubes, though cases have been reported by Sänger (Doran).

Secondary Malignant Disease of the Fallopian Tube.—In cases of malignant tumour of the ovary, the serous coat of the tube is occasionally found to be studded with secondary deposit. Fabricius[3] reports a case of much importance in the interests both of pathological accuracy and of diagnosis. A patient aged forty had a free sanious discharge for several months. This symptom proved due to a small cancerous ovarian tumour, connected with a large hydrosalpinx which adhered to it. Some cancerous growth had perforated the walls of the tube, and floated free inside its dilated canal.

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[1] Sänger, *Centralb. f. Gynäk.* 1886, p. 601.

[2] Janvrin, *Annals of Gynæcology*, Boston, U.S.A., vol. ii.; Charles Dixon Jones, *Amer. Journ. of Obstetrics*, vol. xxviii. 1893.

[3] 'Perforation eines malignen Ovarialtumour in die Tube,' *Wiener Klin. Wochenschrift*, vol. ix. 1896, pp. 59 and 74; and Doran, 'An Unreported Case of Primary Cancer of the Fallopian Tubes in 1847,' *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896.

CHAPTER XI

EXTRA-UTERINE OR ECTOPIC GESTATION.

No apology is needed for placing this subject among others of gynæcological interest; for, although in a way it belongs to obstetrics, so many important factors are under consideration and such great advances have been made in the study of this subject in recent years, that it becomes one of the most important of the diseases of women.

An extra-uterine pregnancy is the result of the permanent arrest of a fertilised ovum in its passage from the ovary to the uterus. Doubtless the normal place for the development of the impregnated ovum is the cavity of the uterus, but it cannot be therefore assumed that the uterus alone is the only possible site of impregnation; and, as spermatozoa may without any doubt invade the tubes normally, fertilisation may occur at any place from the ovary to the uterus. This, possibly, does not frequently occur, but if it does, and the fertilised ovum fails to reach the lumen of the tube, or the tube itself fails to pass it along its calibre towards the uterus, a misplaced or ectopic pregnancy results.

The question naturally arises, what may be the possible causes that may lead to this delay of the oösperm instead of its passing as it should do into the uterus and 'settling' there?[1] Most contradictory and confusing views are held, many of them without any pathological proof, and we have to admit that the causes are still surrounded with obscurity.

Taylor,[2] whose recent work on the subject is worthy of the closest study, says that the fructified ovum should normally soon pass into the uterus, but that the following factors play

[1] Bland-Sutton, *Surgical Diseases of the Ovaries and Fallopian Tubes, including Tubal Pregnancy*, 2nd ed. p. 243, says: 'Extra-uterine pregnancy is the result of active rather than obstructive causes, and when the ovum is fertilised the resulting oösperm engrafts itself at once on the adjacent mucous membrane, whether it be tubal or uterine.'

[2] J. W. Taylor, *Extra-uterine Pregnancy*, 1899.

their part in increasing the tendency towards a tubal, instead of a uterine pregnancy, viz.:—

a. Any want of development of the tube.
b. Any permanent contraction of the tube.
c. Any swelling of the mucous membrane.
d. Any abnormal length of the tube.
e. Any extra weight, or impaired mobility of the ovum at its entrance to tube.
f. Any interference with the peristaltic action of the tube, if this be needed, for propulsion of the ovum.

It is reasonable to conclude that some abnormal arrest of the ovum (whether mechanical or special), in its progress towards the uterus, is the determining factor of a misplaced pregnancy.[1]

Reviewing some of these factors, want of development of the tube is certainly seen in some instances. It is either a congenital condition, if found at a first pregnancy; or it may be acquired, as a sequel of a previous pregnancy from some excess of involution, as from puerperal atrophy.[2] Such tubes are extremely thin, with a wide lumen and few folds, and they exhibit atrophy of muscular structure. Taylor gives in his book references to some of the best known specimens in English museums,[3] which certainly seem to exhibit this condition of **atrophy,** which he thinks may account for the failure of, or interference with, the peristaltic action of the tube to pass the oösperm along to the uterus. In the same way muscular action of a tube may expel the mole of extra-uterine pregnancy into the peritoneum, by a sort of reverse action.

As regards arrest of the oösperm by swelling of the mucosa, much attention has been drawn to this point by Clarence Webster,[4] who describes the occasional swelling and growth in the deeper layers of the tubal mucosa which simulates that of the uterine decidua.

This is not a tubal decidua, for no one has yet been able to detect anything microscopically approaching to the uterine decidua; still, it is possible that such swellings as described by

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[1] Taylor, *op. cit.* p. 15.

[2] Dührssen, *Arch. f. Gyn.* vol. liv. pt. 2.

[3] R. Coll. Surg. 4695; Guy's Hosp. Museum, $2517^{\underline{62}}$, $2517^{\underline{85}}$; St. Thomas's Hosp. Museum, 2484.

[4] Clarence Webster, *Ectopic Pregnancy*, Edinburgh, 1895; and *Tubo-peritoneal Ectopic Gestation*, Edinburgh, 1892.

Clarence Webster may (mechanically) arrest an ovum in its progress down the tube.

Previous inflammation of the tube, or the denuded or granulating surface of its interior, was urged, chiefly by Lawson Tait, as a cause of the retardation of the ovum in the tube; but most recent observers have contradicted this point, and both Taylor and Bland-Sutton have often failed to find any evidence of old salpingitis in tubes removed by operation, and the occlusion of tubes, especially in gonorrhœa and the associated sterility,[1] tell against this theory; in fact, a healthy Fallopian tube is more likely to become pregnant than one that has been inflamed.[2] Still it is possible that in a small proportion of cases chronic salpingitis of a mild type may account for the sterility and subsequent tubal pregnancy, but it fails to account for a very large number of instances; indeed, some of the changes described in such tubes are the consequence, not the cause, of the tubal pregnancy.

Much more rarely the lumen of the tube may be in some way interfered with. Dührssen[3] describes a tubal polypus occluding the uterine end of the tube, again a tubal pregnancy has been found on the outer side of an ovarian tumour or a uterine fibroid (figs. 35, 36, 37), or the tube may be found fixed or stretched by adhesions.

Taylor[4] reports three cases complicated with ovarian tumours, one a dermoid. In all, the tubes were fixed and distorted by the growth of the cysts, or accompanying adhesions.

Although some of the above causes may explain a few of the cases, and the evidence points to some abnormal arrest of the ovum, the causes of tubal pregnancy in a very large number of cases still remains unexplained and undetected.

The age of patients most subject to extra-uterine pregnancy varies. Bland-Sutton's[5] statistics of 100 cases were 20–25 = 10 cases; 25–40 = 86 cases; 40–45 = 4 cases; and, as with uterine pregnancy 25–35 (see chapter on Sterility) may be regarded as the great child-bearing period of life, so the period of functional activity of the uterus represents also the period

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[1] Bland-Sutton, *op. cit.*, paragraph on 'Tubal Pregnancy and Antecedent Sterility,' new ed. 1896, p. 243.

[2] Bland-Sutton, Allbutt and Playfair, p. 453.

[3] Quoted by Taylor, *op. cit.*

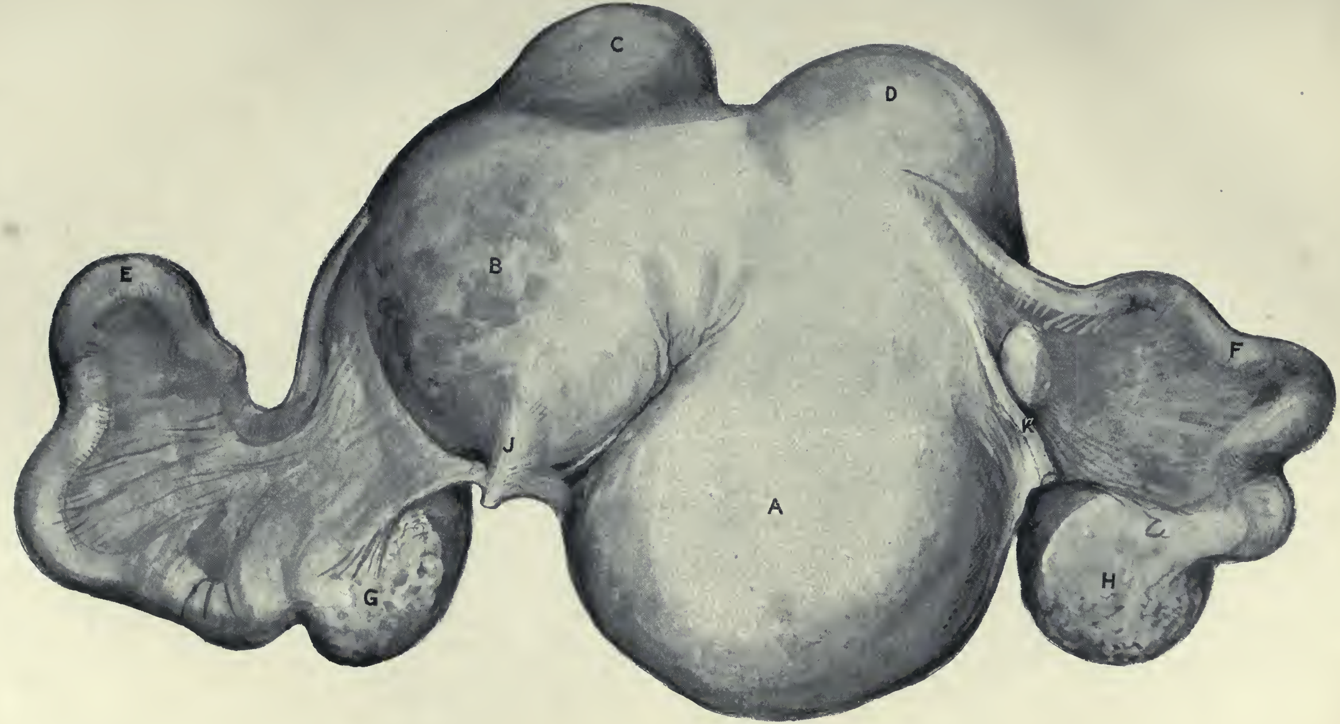
[4] Taylor, *op. cit.* p. 15.

[5] Bland-Sutton, *op. cit.* 1896, p. 244.

Fig. 35.

EARLY ECTOPIC GESTATION (? TUBO-UTERINE) COMPLICATED BY FIBRO-MYOMA OF THE UTERUS.

(View from the front and above.)



A. Interstitial fibro-myoma in anterior wall of uterus.
B and C. Gestation sac filled with blood-clot, forming projections similar in appearance to, but softer in consistence than, those formed by the fibro-myomata.
D. Interstitial fibro-myoma in wall of fundus uteri.
E. Right Fallopian tube (8 inches).
F. Left Fallopian tube (5 inches).
G. Right ovary.
H. Left ovary.
J. Right round ligament, with point of uterine origin displaced outwards by gestation sac.
K. Left round ligament, normal.

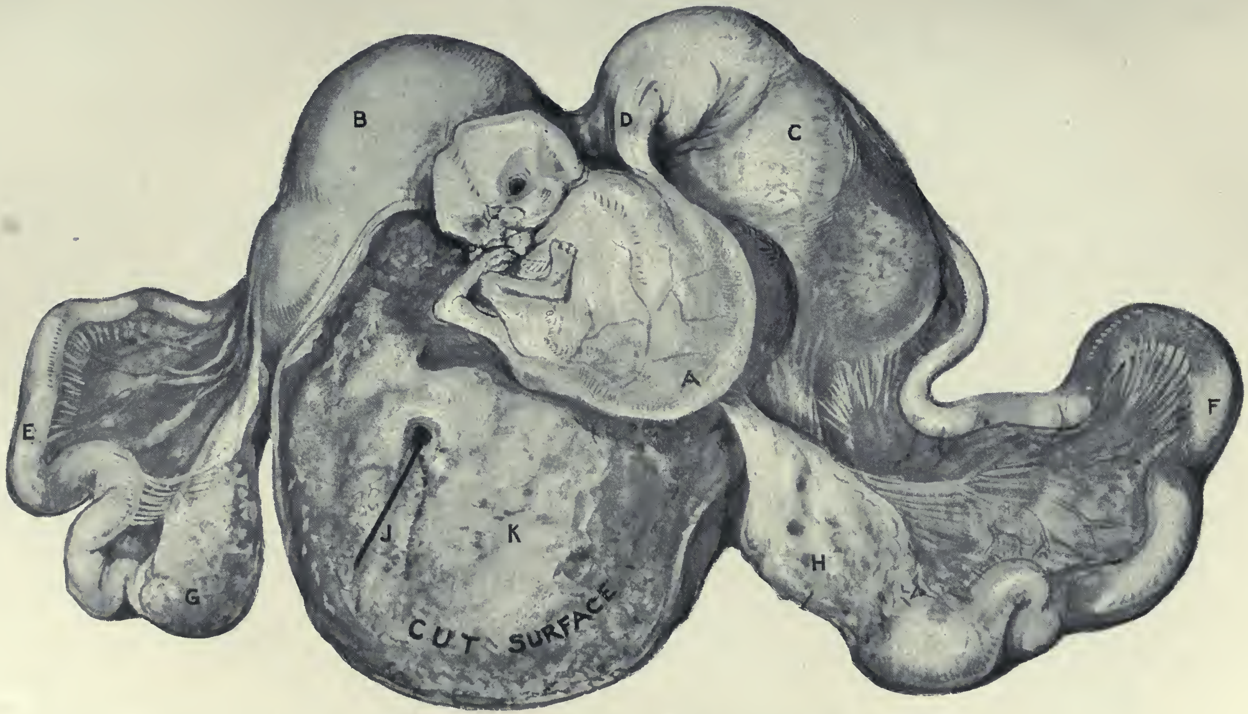
(Cullingworth, 'Trans. Obstet. Soc. Lond.' vol. xl. 1898, plate vii., drawing by R. E. Holding.)

Fig. 36.

EARLY ECTOPIC GESTATION (? TUBO-UTERINE) COMPLICATED BY FIBRO-MYOMA OF THE UTERUS.

(Same Case as Fig. 35.)

(View from below and behind.)

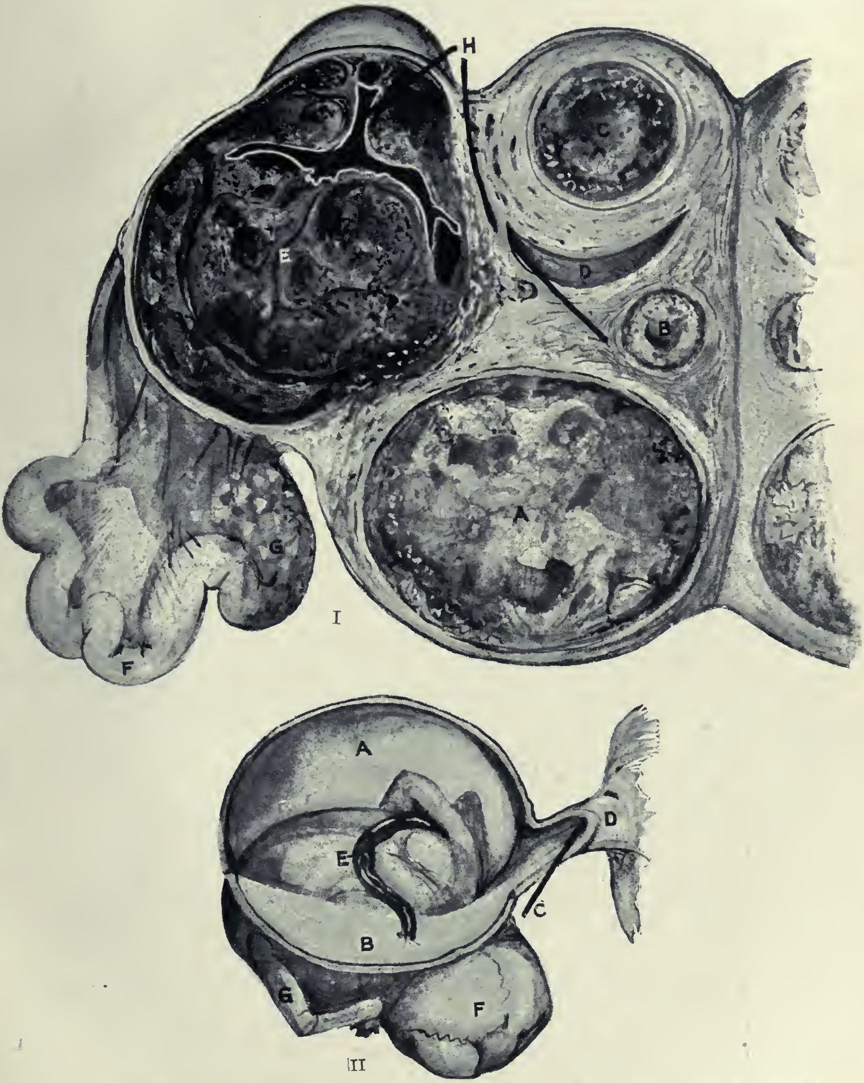


A. Sac, with head and limbs of fœtus protruding from one end found lying in Douglas's pouch.
B. Small interstitial fibro-myoma in posterior wall of uterus.
C. Gestation sac filled with blood-clot.
D. Band of tissue with central canal connecting A with C.
E. Left Fallopian tube.
F. Right Fallopian tube.
G. Left ovary.
H. Right ovary.
J. Bristle passed into uterine canal at junction of body and cervix.
K. Cut surface of uterus, showing plane of division at level of isthmus. Uterine body enlarged by fibro-myoma in anterior wall.

(Cullingworth, 'Trans. Obstet. Soc. Lond.' vol. xl. 1898, plate viii., drawing by R. E. Holding.)

Fig. 37.

EARLY ECTOPIC GESTATION (? TUBO-UTERINE) COMPLICATED BY FIBRO-MYOMA OF THE UTERUS. (Same Case as Figs. 35 and 36.)



I.—*View on Section after hardening.*

A. Fibro-myoma in anterior wall.
B and C. Smaller interstitial fibro-myomata.
D. Cavity of right cornu of uterus laid open, with bristle in uterine aperture of right Fallopian tube.
E. Blood clot filling gestation sac.
F. Right Fallopian tube.
G. Right ovary.
H. Compressed cavity lined by amnion.

II.—*Sac (containing Fœtus) laid open, wall turned down.*

A. Interior of sac.
B. Wall turned down to display interior.
C. Bristle passed through connecting band, showing direct communication with original gestation sac (fig. I. H).
D. Connecting band.
E. Umbilical cord passing across back of fœtus and attached near B to inner wall of sac.
F. Head of fœtus.
G. Limbs of fœtus protruding from sac.

(Cullingworth, 'Trans. Obstet. Soc. Lond.' vol. xl. 1898, plate ix., drawing by R. E. Holding.)

of liability of the tubes to become gravid. It is doubtful if a preceding 'period of sterility' has anything to do with a subsequent gestation outside the uterus, for, as previously stated, this cannot be invariably explained by a previous salpingitis, which, if it occurs, is certainly not the rule. Tubal pregnancy may be the first pregnancy, or it may follow normal pregnancy or an abortion, or there may be an interval after a normal pregnancy, following an extra-uterine one, or a woman may be married for many years and have no pregnancy and then conceive, and such a pregnancy may be extra-uterine. Both tubes may become gravid at the same time, or one tube become pregnant years after the other[1]; tubal may complicate uterine pregnancy, and twin tubal pregnancy is said to occur.[2] A case is recorded where both pregnancies took place in the same tube[3] and the fœtus was well formed in each instance, and had advanced to about three months' growth, the recent fœtus at the operation being living, that of the older pregnancy being a lithopædion. Repeated extra-uterine pregnancy in nearly all cases has of course occurred on the opposite side. It is said to occur in about four per cent. of all cases.[4] Taylor refers in his book to cases quoted by Dührssen and Tait, and gives the history of several others.[5] There appears to be a similarity in such histories; and in some, atrophy or want of development may affect both tubes; in others, the cause found was obviously one-sided, such as a polypus or diverticulum of the tube, some pelvic tumour, or local adhesions.

A curious case is recorded by Cullingworth[6] in which an early extra-uterine pregnancy was complicated by a fibromyoma. (Figs. 35, 36, 37.)

Classification of Varieties.—There is probably one great variety of Ectopic Pregnancy, viz. **Tubal**,[7] all other varieties being later developments either of tubal or of uterine pregnancy, and being caused by secondary invasion from the Fallopian tube or uterus, or some other tissue or organ.

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[1] Lewers, *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 324, 'A Case of Repeated Extra-uterine Pregnancy in the same patient; Laparotomy on each occasion.'

[2] Taylor, *op. cit.* p. 23. [3] Coe, *Amer. Journ. of Obstetrics*, vol. xxvii. p. 855.

[4] Haig Ferguson, 'Repeated Ectopic Pregnancy, *Edin. Med. Journ.* Feb. 1899.

[5] Taylor, *op. cit.* pp. 183–185, 'On Repeated Extra-uterine Pregnancy and its lessons;' Herman, *Brit. Med. Journ.* Sept. 27, 1890, and *Trans. Obstet. Soc. Lond.* vol. xxxii. 1890, p. 307; Galabin, 'Uterine and Tubal Pregnancy co-existing,' *Trans. Obstet. Soc. Lond.* vol. xxiii. 1881, p. 141.

[6] Cullingworth, *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 285; Rumley Dawson, *ibid.* p. 155.

[7] Bland-Sutton, *op. cit.* new ed. 1896, p. 242.

In spite of the hard-and-fast line hitherto taken by writers, that there is only one variety of ectopic gestation, viz. Tubal, of late, increasing evidence has been brought forward of the existence of **Primary Ovarian Pregnancy**, to which some reference must be made.[1]

Martin, Sänger, and Leopold of Leipzig[2] have reported cases which they believed were primarily ovarian, but both were retained fœtations at full term. Leopold's was a lithopædion removed from a woman of seventy-five, thirty-five years after the child had died[3]; here all the original relations of the sac must long ago have been lost, and that part of the ovary enveloped the lithopædion proves little. Howard Kelly[4] admits that he has never seen a case, and says it is the rarest of all forms. He points out that a primary ovarian pregnancy must satisfy Spiegelberg's criteria—viz. the tube must be intact and have no organic connection with the gestation sac, the tumour should be connected with the uterus by the tubo-ovarian ligament, the walls of the sac must contain Graafian follicles in various places, and the albuginea of the ovary must pass directly into the tumour wall. Bland-Sutton[5] points out that a real primary ovarian pregnancy must also be actually contained inside the ovary in a sac in its unruptured membranes; for by the term 'ovarian pregnancy' we mean the fertilisation of an ovum *in situ* before its escape from the follicle.

Patenko[6] reported a case where fœtal bones were contained in a cyst in an ovary which itself was the size of a hen's egg.

In 1898 Catherine Van Tussenbroek[7] reported a curious case at the third Obstetrical and Gynæcological Congress, removed by Kouwer of Harlem in 1893, which she believed to be a real example of ovarian pregnancy.

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[1] *Lancet*, Jan. 12, 1901, pp. 100, 101.

[2] Leopold, *Arch. f. Gyn.* 1892, vol. xix.

[3] Taylor, *op. cit.* p. 177.

[4] H. Kelly, *Operative Gynæcology*, vol. ii. p. 434.

[5] Bland-Sutton, Allbutt and Playfair, *Sys. of Gyn.* p. 472.

[6] For this and other cases see Doran and Beale, *Trans. Obstet. Soc. Lond.* vol. xxxv. 1893, p. 222, and literature.

[7] Van Tussenbroek, *Lancet*, Dec. 29, 1900, p. 1887; *Brit. Med. Journ.* vol. i. 1900, p. 922; *Ann. de Gyn. et d'Obstét.*, 'Un cas de Grossesse ovarienne;' vol. lii. p. 537; and *Semaine Gynéc.* vol. v. Paris, 1899, 1900, p. 34; *Annus Medicus*, 1900.

This case has been further investigated, and Bland-Sutton[1] reported to the Obstetrical Society of London that he had seen the specimen; he showed two of Van Tussenbroek's microscopic sections, which without doubt prove that the condition was really an ovarian pregnancy. The condition is very rare.

The author himself saw the sections, which showed a fœtation with its membranes complete, contained in a follicle in the ovary, with a normal development of syncytium.

Anning and Littlewood,[2] of Leeds, showed at the same meeting a rather similar case from a patient of twenty-eight on whom they had operated for a ruptured extra-uterine gestation. There was a rent in the right ovary, leading to a cavity, which contained some blood; a small ovum, about the size of a Barcelona nut was found amid the blood-clot in the abdomen. The ovum had escaped from the cavity in the ovary, into which it fitted exactly. There was no evidence of rupture in the right tube, which was removed. The left tube was normal, but a cystic portion of the left ovary was also removed. The specimen was shown with microscopic sections of the ovum, sac, and ovarian wall. These two cases appear clearly to demonstrate that primary ovarian pregnancy really exists, and it is an important step in the progress of our knowledge of gynæcological pathology. It behoves us to pay the greatest attention to the further investigation of this point, especially in the careful examination of hæmorrhages and apoplectic extravasations into the ovary, as villi are easily overlooked.

Some of these cases may even be blighted ovarian pregnancies, similar to those that will be described later as Tubal Moles.[3] Hitherto most cases of supposed ovarian pregnancy have been either ruptured tubo-ligamentary pregnancies, or encapsuled hæmatoceles around a gravid tube containing a mole.

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[1] Bland-Sutton, *Trans. Obstet. Soc. Lond.* vol xliii. 1901, part i.; see also *Brit. Med. Journ.* vol. i. 1901, p. 41.

[2] Anning and Littlewood, 'Primary Ovarian Pregnancy, with Rupture fourteen days after last Menstruation,' *Trans. Obstet. Soc. Lond.* vol. xliii., 1901, p. 14; see also Croft, 'An Anomalous Case of Ectopic Gestation, probably Ovarian,' *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 316; *Brit. Med. Journ.* Nov. 17, 1900, p. 1442. This case has been further investigated by Targett, Eden, and the author; it is with very little doubt a genuine case of Primary Ovarian Pregnancy; see Report of Committee on Croft's specimen, *Trans. Obstet. Soc. Lond.* vol. xliii. 1901, p. 24.

[3] See *Brit. Med. Journ.* vol. i. 1901, p. 90, and *Trans. Obstet. Soc. Lond.* vol. xliii. 1891, part. i. p. 19.

Sub-Varieties of Tubal Pregnancy.[1]

- *a.* **Tubo-abdominal,** in which there is secondary invasion of the abdomen (ventral).
- *b.* **Tubo-ligamentary, mesometric, or broad ligament pregnancy,** in which there is secondary invasion of the broad ligament and subperitoneal tissues.
- *c.* **Tubo-uterine or interstitial,** in which there is secondary invasion of the uterus.

Tubal Pregnancy.—This being by far the commonest variety,[2] the changes in the tubes are worthy of close consideration. Certain changes are common to nearly all specimens, the vascularity of the tube is increased, and the ovum is everywhere surrounded by the swollen mucosa; into portions of this the chorionic villi grow, and the ovum becomes attached. The ovarian vessels, and also the uterine, are much increased in size, and there is hyperæmia of the broad ligament. The ovum grows and expands the tube, but there is very little attempt of any compensatory hypertrophy of the tube itself;[3] in fact, the tube is progressively attenuated by distension. Taylor thinks this is either due to the primary thinness of the tube, or to the part of the tube affected; the part near the uterus having a feeble resistance and nutrition, owing to the distribution of the ovarian vessels mainly to the outer positions of the tube. The rapidity of thinning varies; but, as Bland-Sutton says, under normal conditions, Fallopian tubes vary in length and thickness. Distension of the tube gradually effaces the plicæ, and the greatest vascularity affects the abdominal ostium; there is turgescence of the fimbriæ as well as of the muscular and serous coats. The peritoneal margin near the ostium swells and forms a ring around the fimbriæ; sometimes it projects with the muscular coat beyond the fimbriæ, and may even fold over and close them in, thus sealing the tube.[4] This is not always the case, and in many specimens the **abdominal ostium remains wide open** (figs. 42, 46, 49), which is a very important fact, in that it helps us to understand tubal abortion

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[1] Taylor, *op. cit.* p. 16. The groups with variety *a*, 'utero-abdominal pregnancy,' where gestation continues after rupture of the uterus (*ibid.* p. 62).

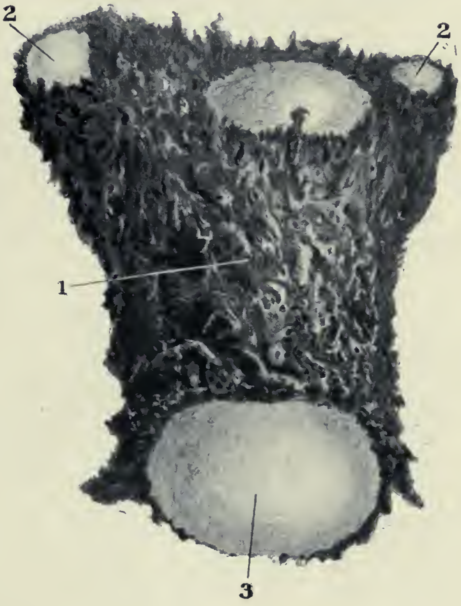
[2] Lawson Tait, *Ectopic Gestation*, 1888, who says it is the only variety.

[3] Taylor, *op. cit.* p. 9.

[4] Sutton, Allbutt and Playfair, p. 454; compare also the salpingitic closure of the tubes in chapter on 'Pelvic Inflammation' in this book; see also, Doran, Allbutt and Playfair, p. 792, fig. 202.

Fig. 38.

A CAST OF THE UTERUS IN EXTRA-UTERINE GESTATION.



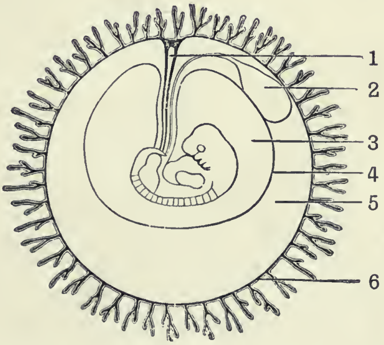
A decidual cast from the uterus in a case of extra-uterine pregnancy. The cast has been distended with cotton wool. Its triangular shape is well shown. Its outer surface is shaggy, and shows the processes of the decidua.

1. Outer surface of cast.
2, 2. Points of entrance of the Fallopian tubes.
3. Cotton wool distending the cast in the region of the internal os uteri.

(Specimen 4694 A. Museum, Royal College of Surgeons.)

Fig. 39.

DIAGRAM OF AN EARLY OVUM TO SHOW THE ARRANGEMENT OF THE MEMBRANES.



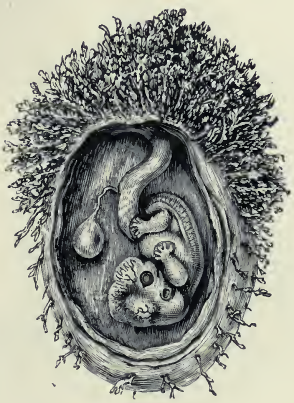
1. Allantois.
2. Umbilical vesicle.
3. Amniotic cavity.
4. Amnion.
5. Subchorionic chamber.*
6. Chorion with villi.

(Bland-Sutton, ' Trans. Obstet. Soc. Lond.' vol. xxxvi. 1894, p. 197.)

* In tubal mole blood is extravasated into this space from the ovuline vessels. The red corpuscles are nucleated, and the white cells especially numerous.

Fig. 40.

AN EARLY TUBAL OVUM.



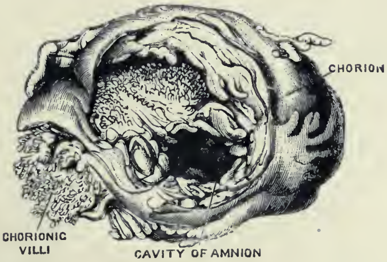
An ovum from a case of ruptured extra-uterine pregnancy at about the sixth week (natural size). The ovum was lying in the Fallopian tube, which had ruptured, and the peritoneal cavity was full of blood at the time of operation.

The specimen shows the space between the chorion and amnion (subchorionic chamber), and the polar disposition of the chorionic villi.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxvi. 1894, p. 197.)

Fig. 41.

A TUBAL MOLE. (Natural Size.)

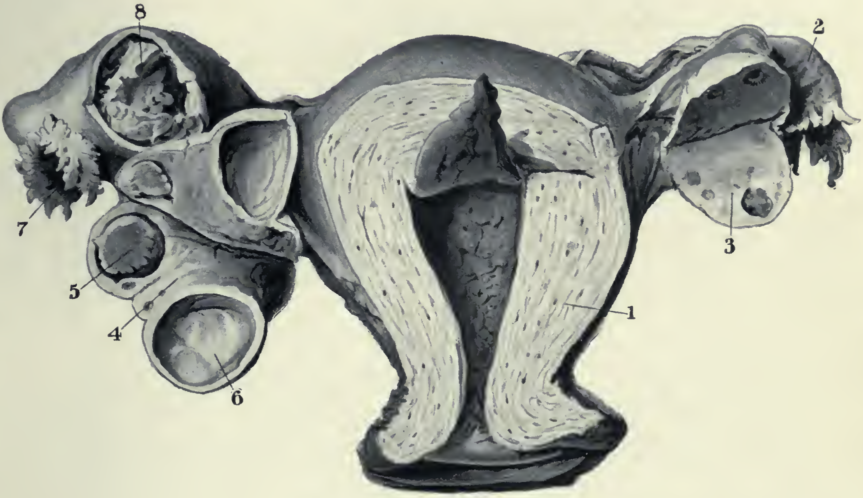


Showing the space between the amnion and chorion (the subchorionic chamber). In tubal mole hæmorrhage takes place into this from the vessels of the ovum. This explains the elliptic shape of the ovum as seen in the above drawing, for if the effusion of blood happens at the time this space is large the amnion will be squeezed to one pole of the ovum. The blood is limited by the two membranes, of which the inner one is the amnion and the outer the chorion.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxvi. 1894, p. 198.)

Fig. 42.

EXTRA-UTERINE GESTATION, SHOWING THE FORMATION OF A TUBAL MOLE.



A uterus and tubes from a case of extra-uterine pregnancy. The left tube contains an early tubal mole (8). The tube has been opened to show the clot in its interior. The oval shape of the mole is well shown. It is to be noted that the orifice of the left Fallopian tube is wide open.

A well-marked decidual cast, which is turned upwards, is seen in the uterus.

1. The uterus, which is considerably enlarged.
2. Fallopian tube of the right side.
3. Right ovary.
4. Ovary of left side, containing a cyst (6) and a recent corpus luteum (5).
7. The open orifice of the left Fallopian tube.
8. A tubal mole occupying the left Fallopian tube, which has been opened to show the clot in its interior.

(Specimen 3071. Museum, St. Bartholomew's Hospital.)

and the leaking of blood from the open end of a Fallopian tube containing a mole, causing the 'blood-drip,' which is the frequent source of Pelvic Hæmatocele.

Still, it does not follow that a tube even with an open end may not rupture, though a gravid tube with an occluded ostium invariably bursts.

Another important change is the **increase in size of the uterus** while pregnancy is going on in the tube. (Figs. 42, 53, 57.)

Together with this is the **formation of the uterine decidua**, which invariably occurs with extra-uterine gestation (figs. 38–42), and we believe that the decidual *membrane* is always the result of pregnancy, though decidual cells mean nothing. Some curious cases of '**Spurious Abortion**'[1] have occurred in which casts of the uterus do appear to have been passed apart from pregnancy, either intra- or extra-uterine, and having no connection with dysmenorrhœa; if so, it is no longer possible to regard the discharge of a decidual cast as pathognomonic of extra-uterine pregnancy.

In all extra-uterine cases, death of the ovum, or rupture of the tube, is associated with the shedding of the decidua, which may be passed entire or in shreds. In some cases the decidua may be retained for weeks after rupture, or even after operation at full time.[2]

The **decidua** in extra-uterine pregnancy may form a perfect cast of the uterus, with three openings for the two Fallopian tubes and the cervix. The outer aspect is rough (fig. 38), and the inner smooth, and dotted with the orifices of the uterine glands. So far as is known, a decidua never forms in the tubes or cervical canal. Its detachment begins at the os internum. The membrane consists of decidual, *i.e.* uterine connective tissue cells, and is in the first month about $\frac{2}{5}$ inch thick, fleshy, ovoid, and vascular, and corresponds to the enlarged cavity of the uterus. It can hardly be confounded with the thin delicate cast of the uterus in membranous dysmenorrhœa, which averages $\frac{1}{25}$–$\frac{1}{12}$ inch thick.[3] Any membrane approaching $\frac{1}{4}$ inch in thickness cannot be a dysmenorrhœal membrane unless additionally diseased. The length of the sac in dysmenorrhœa is only $1\frac{3}{4}$–2 inches, while extra-uterine or abortion sacs are always much larger.

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[1] Eden, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 132; Griffith, *ibid.* vol. xxxvi. 1894, p. 335; Dakin, *ibid.* vol. xxxviii. 1896, p. 385.

[2] Taylor, *op. cit.* p. 19.

[3] Champneys, *Painful Menstruation*, p. 53.

In considering the abortion sac of intra-uterine pregnancy, search must be made for placental tissues, especially villi; their absence is a matter very difficult to prove, their presence is often plain. It would not, however, be safe to assert [1] that a sac was not the product of conception because no villi could be found, though it would be an argument against it certainly; and the greatest patience may have to be observed in cutting large numbers of serial sections of suspected tissue in the search for villi. The greatest interest attaches to Griffiths', Dakin's, and Eden's [2] cases of casts passed from the uterus, in which there was no evidence of gestation, in relation to this matter.

The **decidua in cases of double uterus** is important, for if pregnancy occurs in one half of a bicornute uterus, a decidua forms in the unimpregnated cornu (fig. 57). Bland-Sutton [3] has observed the same thing in animals normally possessing bicornute uteri (ungulates and lemurs).

Other membranous structures likely to be confounded with the above [4] are fibrin, blood-clot, coagulated mucus, casts of vagina, casts of the bladder, exfoliation of uterine mucous membrane in cases of phosphorus poisoning and cholera, and, lastly, foreign bodies.

Tubal Pregnancy. Early Rupture.—This generally takes place between the second and sixth week, the first warning being the sudden and copious bleeding; even the history of pregnancy may be wanting, or only represented by the account of menstruation being missed a week or less. Death may rapidly ensue, even in $7\frac{1}{2}$ hours (Tait), generally in twelve to forty-eight hours, unless something be done. Very little compensatory hypertrophy of the tube is ever found in these cases; in fact, in some cases atrophy is described (Taylor). At all events, the most remarkable fact is the terrific amount of intra-peritoneal bleeding from apparently so small a cause, the hæmorrhage being diffuse, and with no tendency to any limitation.

In a large number of cases pregnancy in the Fallopian tube

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[1] Champneys, *op. cit.* p. 51.

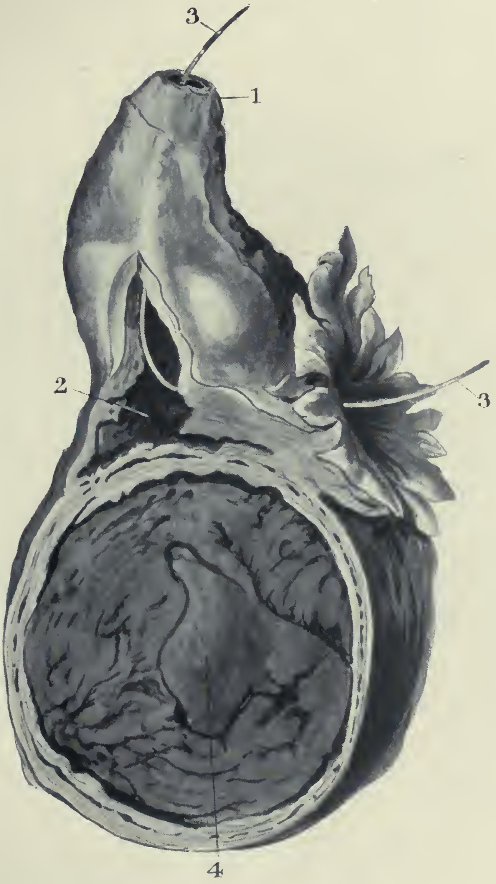
[2] Eden, *op. cit.*

[3] Allbutt and Playfair, p. 465.

[4] Champneys, *op. cit.* p. 48.

FIG. 43.

SACCULATED HÆMATOSALPINX, PROBABLY DUE TO TUBAL GESTATION.



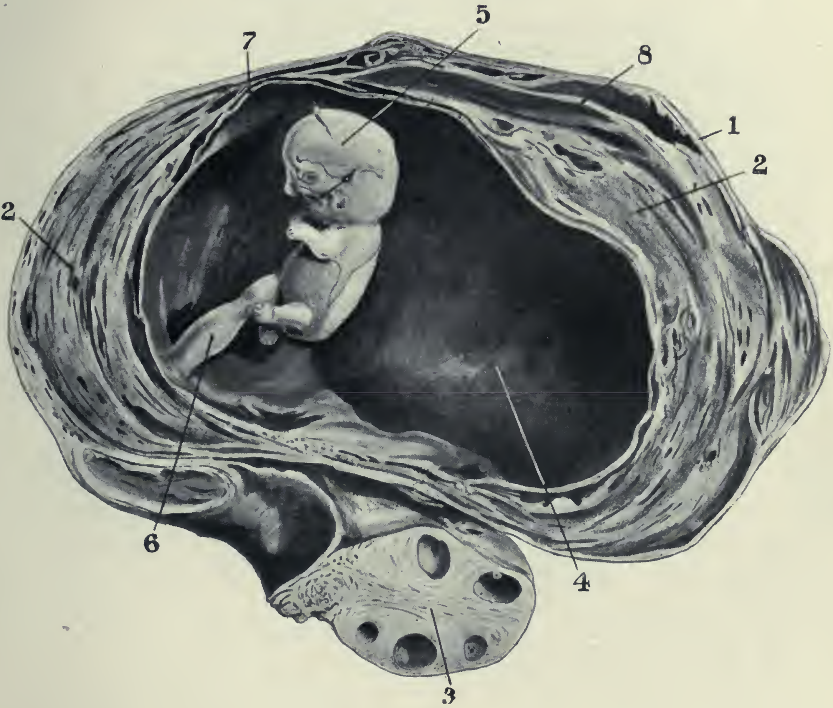
A Fallopian tube; in connection with its distal end is a closed cyst-like cavity full of blood-clot. It is quite separate from the tube. It was probably the result of an extra-uterine pregnancy with formation of a tubal mole, but no villi could be found in the clot.

1. Uterine end of Fallopian tube.
2. Oblique section through the tube to show the bristle (3).
4. Blood-clot in a cystic cavity which does not communicate with the tube.

(Specimen 2480. Museum, St. Thomas's Hospital. Cullingworth, 'Trans. Obstet. Soc. Lond.' vol. xxxiv. 1892, p. 182; *id.* 'St. Thomas's Hosp. Reports,' vol. xxi. p. 42; *id.* 'Clinical Illustrations of Diseases of the Fallopian Tubes,' plate xi. fig. 1.)

FIG. 44.

A TUBAL MOLE.



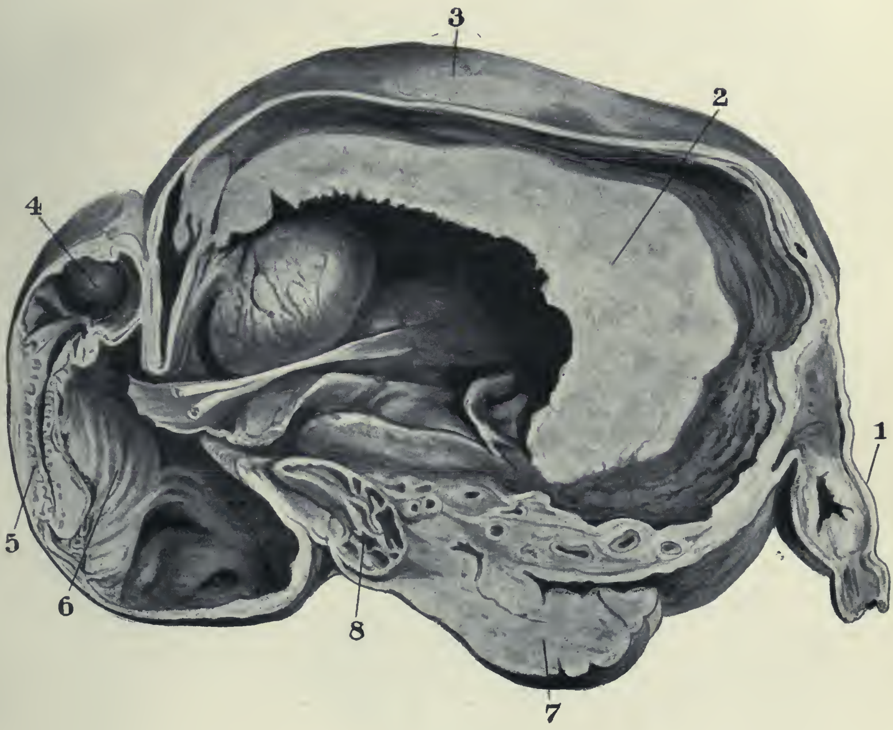
Section of a Fallopian tube showing a tubal mole in its interior. It contains a fœtus, which is in perfect preservation.

1. Wall of tube.
2, 2. Layers of laminated clot between the amnion and chorion (*i.e.* in the subchorionic chamber).
3. Ovary, occupied by several cystic follicles.
4. Cavity of amnion.
5. Fœtus in perfect preservation, measuring ¾ inch in length.
6. Umbilical cord.
7. Amnion.
8. Chorion.

(Doran, 'Trans. Obstet. Soc. Lond.' vol. xlii. 1900, p. 134.)

Fig. 45.

UNRUPTURED TUBAL GESTATION WITH APOPLEXY OF THE OVUM.



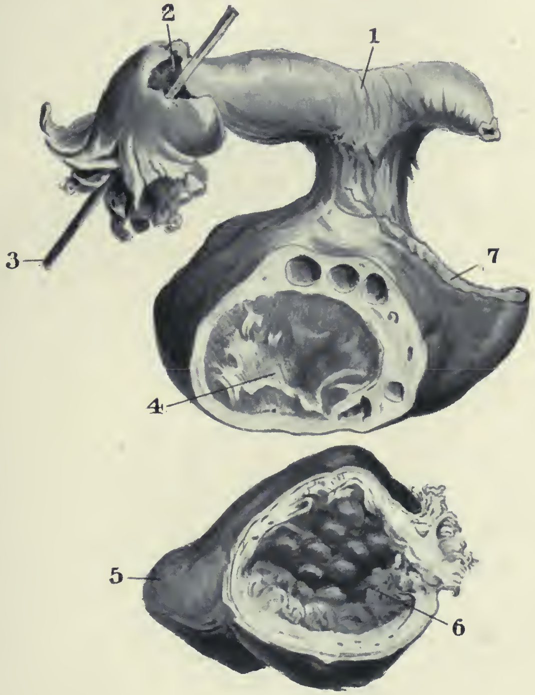
Longitudinal section of a Fallopian tube, the greater part of which is occupied by a mass of pale clot. To the left of the specimen is a pouch in the wall of the tube communicating with the main cavity. Against the walls of this pouch lies a fœtus (4, 5, 6) compressed by blood-clot. The amnial cavity and umbilical cord are seen in the centre of the tube. The cranial cavity and vertebral column of the fœtus are seen in section.

1. Uterine end of tube cut obliquely.
2. Blood-clot, the outer layers of which are laminated; the inner are pale. In the centre of this is the amnial cavity and umbilical cord (which should not be represented as dividing). In places masses of clot are bulging inwards towards the amnial cavity.
3. Main portion of tube.
4. Brain cavity of fœtus.
5. Vertebral column.
6. Ribs.
7. Ovary.
8. Cyst in the ovary.

(Specimen 2481. Museum, St. Thomas's Hospital. Cullingworth, 'Clinical Illustrations of Diseases of the Fallopian Tubes,' p. 52, plate xii.; *id.* 'St. Thomas's Hosp. Reports,' vol. xxi.)

Fig. 46.

FALLOPIAN TUBE, OVARY, AND TUBAL MOLE
(EXTRA-UTERINE GESTATION).



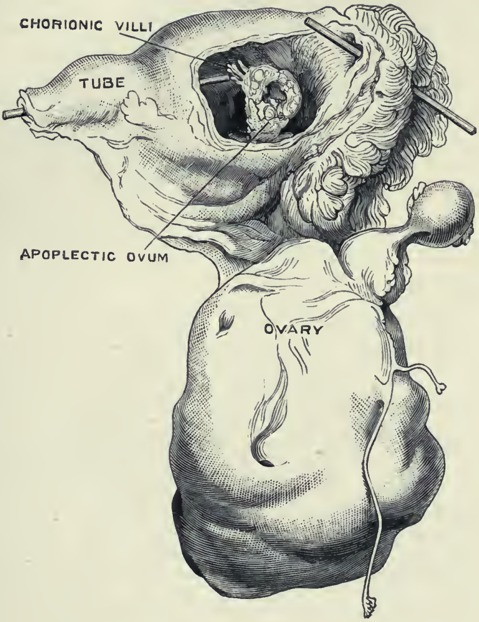
The upper drawing shows the Fallopian tube and ovary. The ovary contains a large corpus luteum. The lower drawing is the tubal mole which has escaped from the Fallopian tube and was found lying among the fimbriæ. A rupture in the tube is shown (2).

1. Fallopian tube.
2. A rupture in the tube. A glass rod (3) has been passed from this through the open end of the tube.
4. A large corpus luteum in the ovary.
5. The tubal mole which has escaped from the tube. It exhibits the typical eccentrically ovoid shape; its outer envelope is the chorion. Villi are seen projecting on the right-hand border.
6. Clot in the interior, occupying the subchorionic chamber.
7. Cut surface of ovary in region of pedicle.

(Specimen 4695 Museum, Royal College of Surgeons. Bland-Sutton, Jacksonian Essay, 1892.)

Fig. 47.

TUBAL ABORTION.



A Fallopian tube opened to expose an apoplectic ovum in tubal pregnancy. The ampulla is dilated to the thickness of the thumb; its ostium is wide and patent. A rounded firm body is seen adherent to the tubal mucous membrane; it is covered at one place by villi. Sections showed this body to be an ovum with blood extravasated into its membranes.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxii. 1890, p. 344.)

[The ovary and tube of the other side are shown in fig. 23.]

leads to the formation of a **Tubal Mole**[1] (figs. 40, 41, 42). Blood is poured out into the subchorionic chamber (figs. 39, 40) of the ovum, between the amnion and chorion, from the circulation of the embryo (figs. 39, 40, 41), which is thus injured or destroyed. A mole resembles a firm blood-clot in colour and consistence; it is ovoid in shape (figs. 41, 42). On dissecting it a cavity is found containing fluid, the walls of which are smooth and lined with amnion (fig. 44); an embryo (figs. 44, 45) or traces of it may be found, or the embryo may have disappeared. The amniotic cavity usually occurs at one end of the mole; outside this is clot, covering which, and forming the envelope of the mole,[2] is the chorion, and villi can be demonstrated. The blood-clot between amnion and chorion is without doubt furnished by the embryo, and not the mother, since the corpuscles are found nucleated. Of course, blood-clot found on the surface of the chorion of the mole is maternal, but this does not always occur.

Another very important point is the occurrence of **Tubal Abortion** (figs. 46, 47). It is not as common as tubal mole, since the attachment of a mole to the tube is often very firm, but the nearer an ovum lies to the abdominal end of the tube (provided its orifice remains open[3] [figs. 42, 46, 49], as it may do), the greater is the chance that such an ovum may be expelled into the abdominal cavity; this is called tubal abortion. The ovum is nearly always converted previously into a mole. It is discharged into the peritoneal cavity with a more or less copious hæmorrhage, which may be fatal. At operation no rupture of the tube may be found, and the mole often escapes notice amid the clot. At other times the mole remains attached in the mouth of the tube (fig. 40); it is a constant source of hyperæmia, and is the cause of repeated and dangerous bleedings into the abdominal cavity. The weight of the mole in the tube frequently drags the tube down behind the uterus, where it gets fixed, and a complex mass of tube, ovary, broad ligament, blood-clot, and adhesions results.

The great importance of these cases of tubal mole is their liability to cause bleeding into the peritoneum (Intra-peritoneal Hæmatocele), which is generally due to a constant leaking or 'blood-drip' rather than to a profuse sudden bleeding.[1]

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[1] Bland-Sutton, article, 'Extra-uterine Gestation,' Allbutt and Playfair, p. 455, and *id. Surgical Diseases of the Ovaries and Fallopian Tubes*, 2nd ed. 1896, p. 251.

[2] See Doran, 'Tubal Mole,' *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 135, plate xii. See fig. 44 in this book.

[3] Cullingworth, *Clinical Illustrations of Diseases of Fallopian Tubes*, plate ix. fig. 2, p. 32.

Intra-peritoneal Hæmatocele in women is almost always the result of tubal pregnancy;[2] sometimes it is due to rupture, but far oftener to bleeding from the unruptured tube containing a mole[3] (figs. 48, 49). In this case the bleeding, though almost continuous or frequently repeated, is moderate in amount, and a well defined tumour (hæmatocele) is the result. This occupies the pelvis at first, in contact with tube, ovary, and back of uterus; as it grows it rises into the abdomen, and comes in contact with intestine and omentum, adhesions from which now roof it in above; but it is important to remember that the tumour is intra-peritoneal, and is at some point in contact with the open mouth of the Fallopian tube. The tumour may grow by repeated hæmorrhage, and become a definite abdominal mass, which displaces the uterus forwards and upwards, dragging the bladder with it. This position of the uterus is most important in distinguishing the condition from retroversion of the gravid uterus. If fresh bleeding does not occur from the tube the hæmatocele may cease to grow, and it becomes encapsuled by adhesions and alterations in the outer layers of the blood-clots, forming a sort of spurious cyst.[4] It may even be gradually absorbed; or suppuration may occur.

According to Taylor,[5] the following **varieties of Hæmatocele** occur:

a. The **well defined,** due to bleeding from a mole-containing tube; the true intra-peritoneal form.

b. The **diffuse,** from rupture of a gravid tube. This is really not Hæmatocele at all; it is diffuse hæmorrhage.

c. The **unstable,** growing Hæmatocele, in which the

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[1] Bland-Sutton, *Diseases of Ovaries and Fallopian Tubes*, 2nd ed. p. 257, 'On the Causes of Pelvic Hæmatocele.'

[2] Taylor, *op. cit.* p. 33.

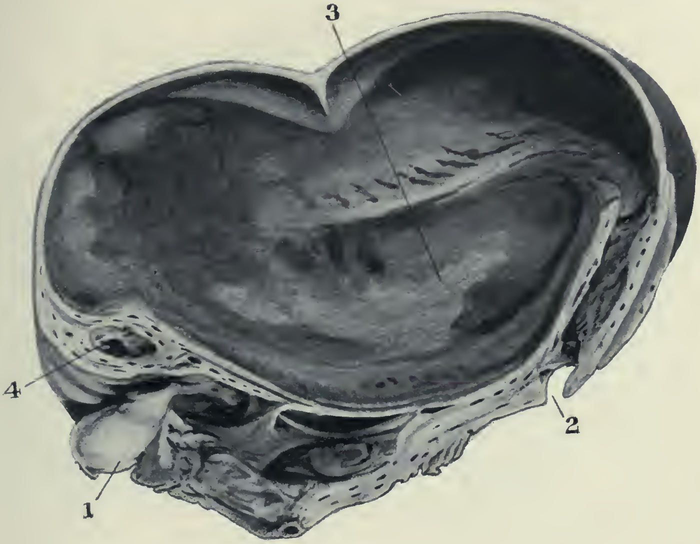
[3] Cullingworth, address to Oxford Med. Soc. Nov. 12, 1897, 'Hæmatocele considered from a modern standpoint,' *Lancet*, vol. i. 1897, p. 1662; *id.* 'Discussion on Intra-peritoneal Hæmatocele,' *Brit. Gyn. Journ.* 1894; *id. Trans. Obstet. Soc. Lond.* vol. xxxi. 1889, p. 226; *id. Diseases of Fallopian Tubes*, plate ix. figs. 1, 2; *id. St. Thomas's Hospital Reports*, vol. xxi.; see also article in present book on 'Hæmatosalpinx.'

[4] Bland-Sutton, *op. cit.* 1896, p. 266.

[5] Taylor, *op. cit.* p. 34.

Fig. 48.

HÆMATOSALPINX PROBABLY DUE TO EXTRA-UTERINE GESTATION.



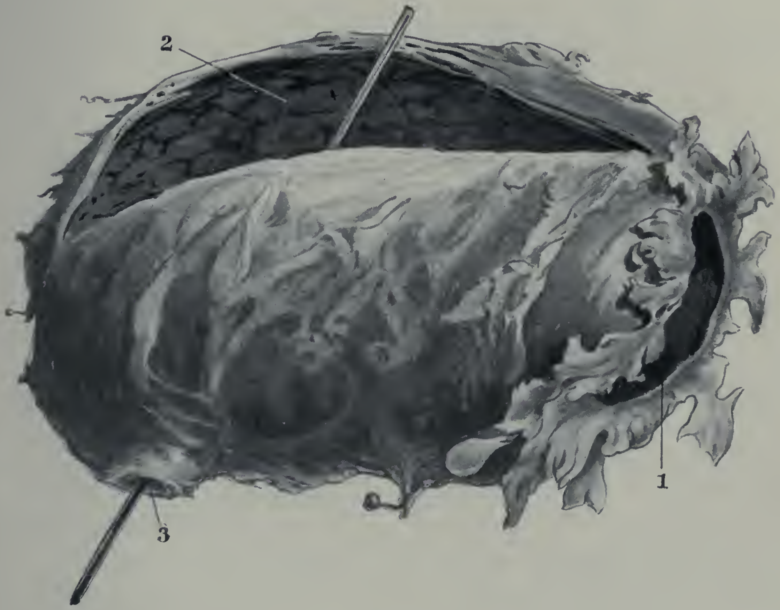
Longitudinal section of a Fallopian tube which was distended with blood-clot. A portion of the fimbriated extremity is seen, it is sufficiently open to admit a goose-quill; through it blood had escaped into the peritoneal cavity (hæmatocele). The clinical history pointed to tubal gestation, but no organised tissue was detected in the clot or the hæmatocele.

1. Cut end of uterine end of Fallopian tube.
2. Fimbriated end of tube.
3. Cavity of the dilated tube from which the clot has been removed. No villi were found in the clot.
4. Section of tube near uterine end.

(Specimen 2478. Museum, St. Thomas's Hospital. Cullingworth, 'Clinical Illustrations of Diseases of the Fallopian Tubes,' plate ix. fig. 1; *id.* 'St. Thomas's Hosp. Reports,' vol. xxi.)

Fig. 49.

FALLOPIAN TUBE DISTENDED WITH BLOOD-CLOT, PROBABLY DUE TO EXTRA-UTERINE GESTATION.



A Fallopian tube (3 inches by 2 inches) distended by blood-clot. The abdominal ostium is wide open, and blood-clot was hanging from it at the time of operation. The tube was surrounded by a large intra-peritoneal hæmatocele (30 ounces of clot). The clinical history pointed strongly to extra-uterine gestation, but no villi were found.

1. Widely dilated fimbriated extremity. (Diameter of ostium = 1 inch.)
2. Main cavity of tube occupied by clot. The surface of the tube is covered with firm clot deposited from the hæmatocele.
3. Glass rod passed through uterine end of tube.

(Specimen 2479. Museum, St. Thomas's Hospital. Cullingworth, 'Clinical Illustrations of Diseases of the Fallopian Tubes,' plate ix. fig. 2; *id.* 'Trans. Obstet. Soc. Lond.' 1889, p. 226.)

tumour formed by rupture is subject to violent and sudden alterations from fresh bleeding: the resulting tumour occupying a position midway between intraperitoneal hæmatocele and diffuse hæmorrhage.

Later Rupture of the Pregnant Tube.—If early rupture of the tube or the other processes previously described do not take place, the pregnancy may go on, but later rupture is sure to take place; generally from the second to the fourth month.

This may occur:

a. Upwards into the peritoneum.
b. Downwards into the cellular tissue of the broad ligament.

a. **Rupture upwards into the Peritoneum.**—This may or may not be accompanied with severe hæmorrhage; much depends on how much of the placenta be separated from the tube during the process of extension. In most cases there is hæmorrhage and the formation of a hæmatocele, but it is an unstable, ever increasing tumour; encapsulation is rare. It is larger at its onset and rapidly increases in size,[1] but this may not necessarily occur, and granted the placenta still retains its connection with the tube, which it practically always does, then the fœtus escapes in its unruptured amnion into the abdominal cavity and continues to grow, forming the subvariety of Tubal Pregnancy called **Tubo-abdominal.** This is the ventral or abdominal form of other writers. Such cases may go to term.

Taylor[2] is the authority quoted on this matter, which is contrary to the opinions held by Tait and Bland-Sutton[3], as up to the present it has been taught that every extra-uterine pregnancy which has survived primary rupture and has continued its development has done so beneath the peritoneum, or mainly in a sac formed by the layers of the broad ligament, and that fœtuses found 'free' amid the intestines at full time were due to late rupture of this broad ligament variety.[4]

Certainly Taylor brings forward some very interesting evidence on the point of Tubo-abdominal pregnancy, which is entitled to close consideration. In such cases at term, the fœtus though apparently free[1] in the abdomen, was found to be really enclosed in the unruptured amnion, which could be traced from coil to coil of intestine[2] completing the sac in which the pregnancy had developed. Others have reported somewhat similar cases at term. The placenta in these cases always remains fixed, growing mainly or entirely from the interior, or from spread-out remnants, of the tube; or if not from the tube, the placenta is in the upper wall of a broad ligament sac, which has ruptured to allow the fœtus to escape. Taylor[3] differentiates four different relations of the placenta to the main gestation sac in abdominal pregnancy, and points out that the sac in true tubo-abdominal pregnancy is always extremely thin and easily broken; if completely independent it may admit of removal with the child, but it is extremely doubtful if it could ever possess sufficient independence and consistency to admit of any suture at the time of operation.

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[1] Taylor, *op. cit.* p. 43.

[2] Taylor, *op. cit.* p. 47.

[3] Bland-Sutton, *op. cit.* 2nd ed. p. 268; Lawson Tait, *Lectures on Ectopic Pregnancy*, 1888, p. 32.

[4] Tait, *op. cit.*

b. **Tubo-Ligamentary, or Broad Ligament Pregnancy.**[4]—This is due to the extension of the pregnancy downwards between the layers of the mesosalpinx. It can only occur at the middle portion of the tube. As the tube expands it separates the layers and grows downwards into the connective tissue space beneath, and is therefore in the broad ligament and beneath the peritoneum; this is accompanied by hæmorrhage, and a broad ligament **Hæmatoma** results. This may be the end of the condition, the pregnancy is stopped, and the blood may be slowly absorbed, causing no subsequent trouble; or the pregnancy may continue in this situation.[5]

Broad Ligament Hæmatoma.—Unlike retro-peritoneal hæmatocele, hæmatoma is only in the minority of cases due to extra-uterine pregnancy. It may be the sequel of an operation, especially if the broad ligament veins be punctured with

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[1] Bland-Sutton, *op. cit.*, 2nd ed. 1896, describes the fœtus in some cases as being free among the bowels (p. 278), 'disporting itself among the intestines' (p. 277), due to secondary intra-peritoneal rupture.

[2] Taylor, 'Second Case of Abdominal Pregnancy,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 178, and *ibid.* vol. xxxiii. p. 115; Champneys, 'Primary Laparotomy in Cases of Extra-uterine Pregnancy' *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 456; see also Clarence Webster, *Ectopic Pregnancy*; Lawson Tait, *Trans. Obstet. Soc. Lond.* vol. xxxiv. 1892, p. 192; Jessop, *Trans. Obstet. Soc. Lond.* vol. xviii. 1876, p. 261.

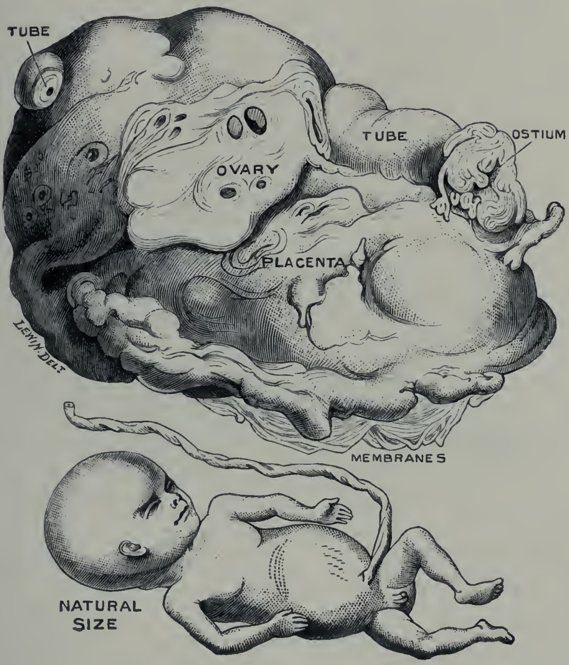
[3] Taylor, *op. cit.* p. 59.

[4] Parry, *Extra-uterine Pregnancy*, London, 1876.

[5] Bland-Sutton, article, 'Extra-uterine Gestation,' Allbutt and Playfair, p. 463.

Fig. 50.

TUBAL PREGNANCY. RUPTURE DOWNWARDS INTO THE BROAD LIGAMENT.

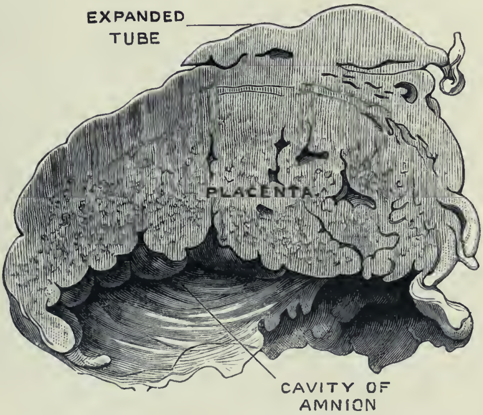


The gestation sac and embryo from a case of tubal pregnancy at about the twelfth week. The sac consists of the Fallopian tube, ovary, and adjacent portion of the broad ligament between which the ovum had escaped and grown. Secondary rupture of this sac in the broad ligament led to escape of blood into the peritoneum. The placenta was situated above the embryo. The tube is partially closed at the abdominal end.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxiii. 1891, p. 72.)

Fig. 51.

TUBAL PREGNANCY.

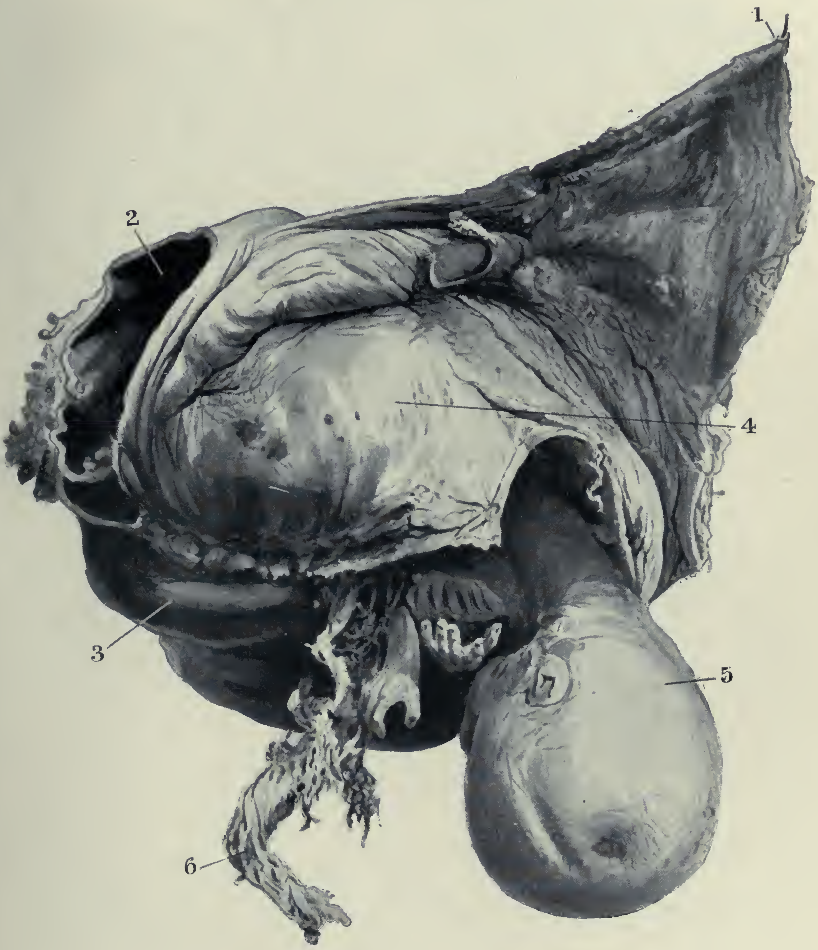


Portion of the placenta from the same case of tubal pregnancy shown in fig. 50. It had ruptured downwards between the layers of the broad ligament. A transverse section has been made of the gestation sac to show the cut surface of the placenta, which was situated above the embryo.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxiii. 1891, p. 72. See also Berry Hart, 'Minute Anatomy of the Placenta in Extra-Uterine Gestation,' 'Selected Papers in Gynæcology and Obstetrics,' Edin. 1893, p. 157.)

Fig. 52.

EXTRA-UTERINE FŒTATION. RUPTURE OF AN INTRA-LIGAMENTOUS PREGNANCY AT THE THIRD MONTH.

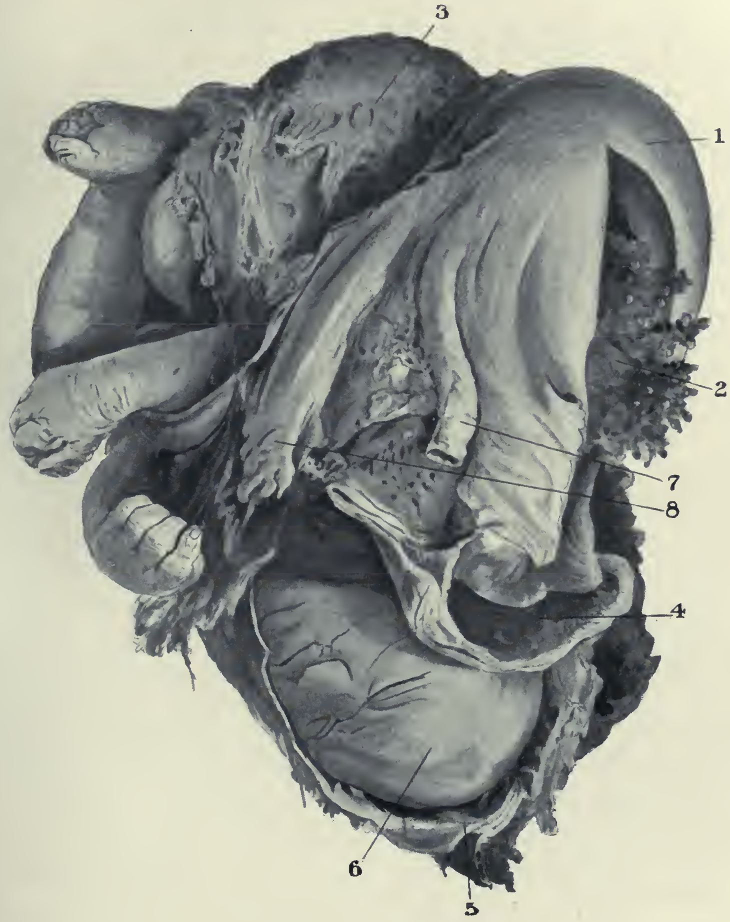


1. Uterine end of Fallopian tube.
2. The fimbriated extremity of the Fallopian tube, which has been opened. It contained clot.
3. Remains of ovary.
4. The broad-ligament sac, which has ruptured at its lower part.
5. Fœtus (at about the third month) projecting through the rupture.
6. Portions of decidua and placenta.

(Specimen 4695 G. Museum, Royal College of Surgeons. Doran, 'Lancet,' vol. i. March 28, 1896, p. 836.)

FIG. 53.

EXTRA-UTERINE GESTATION (NEARLY FULL TERM).



The uterus, fœtus, and membranes of an extra-uterine gestation removed post mortem from a woman who died suddenly at about the eighth month of pregnancy after examination at a medical consultation, when the condition was diagnosed as malignant ovarian disease. The fœtus lies in Douglas's pouch. The rectum is closely adherent to the back of the sac containing the fœtus. The uterus is 5 inches long and contains a well-formed decidua. At the upper part, remains of the sac and placenta are seen. The right Fallopian tube forms part of the sac.

1. Much-enlarged uterus containing a decidua.
2. Decidua.
3. Upper part of sac and placenta.
4. Vagina.
5. Douglas's pouch.
6. Fœtus at about the eighth month.
7. Round ligament.
8. Fallopian tube of right side.

Specimen 4697. Museum, Royal College of Surgeons. Doran, in Discussion on Lawson Tait's paper 'On a Second Successful Case of Gastrotomy in Extra-Uterine Pregnancy,' 'Brit. Med. Journ.' vol. ii. 1879, p. 779; 'Lancet,' vol. ii. 1879, p. 731.)

instruments, or it may occur after labour or abortion, or with ruptures or tears of the uterus.

Hæmatoma of the broad ligament in connection with extra-uterine pregnancy is of sudden onset: it is distinctly lateral, very tense, fixed, and hard; it depresses the fornix of the vagina on that side, and above may be felt near Poupart's ligament. The blood is under tension, and may cause great pain, which may be paroxysmal.[1] It may even surround the rectum in front, or constrict it by tension on the fascia, as in parametritis.

Further Course of Tubo-ligamentary Pregnancy.—After rupture of the tube downwards the fœtus may continue to grow beneath the peritoneum in a sac formed by the expanded layers of the broad ligament, the pregnancy being roofed in as well by the expanded ruptured tube, to which the placenta is still attached (figs. 50, 51). As the fœtus grows, the placenta, being above it, is a constant source of danger, for either the case must end in secondary rupture (fig. 52)—a most serious matter—or the pelvic peritoneum must be continually displaced by the growing fœtus[2] (fig. 53).

Berry Hart's sectional anatomy[3] on this subject should be consulted, and is of the greatest importance.

This displacement of the peritoneum, and the consequent risks to the placenta, is a constant danger in tubo-ligamentary pregnancies which are growing.

Taylor points out that the **peritoneal displacement** in such cases varies, and may be—

a. **Anterior-ligamentary,** the peritoneum being raised from the anterior abdominal wall and bladder, the pregnancy being sub-peritoneal and close behind the abdominal wall. It is also called **sub-peritoneo-pelvic or sub-peritoneo-abdominal.**

b. **Posterior-ligamentary,** or retro-peritoneal, which Taylor[4] and Dunning,[5] speaking from operative evidence, say is commoner than the anterior. In this case the peritoneum is raised upwards from the posterior pelvic wall and rectum as far as the sacral promontory, the anterior peritoneum being undisturbed; thus the tumour looks like a 'pseudo-uterus' at operation, till its posterior relations are explored.[1]

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[1] Cripps, *Brit. Med. Journ.* vol. i. 1896, p. 780.

[2] Taylor, *Extra-uterine Pregnancy*, p. 68.

[3] Berry Hart, *Selected Papers in Gynæcology and Obstetrics*, Edin. and Lond. 1893, Chaps. 17, 18, 19.

[4] Taylor, *op. cit.* p. 77.

[5] Dunning, *Amer. Journ. Obstetrics*, July, 1897.

The **displacement of the placenta** is sometimes remarkable: it generally lies above the child (fig. 53) in tubo-ligamentary pregnancy. It is rare for the placenta to be depressed beneath the fœtus; later the child may pass either behind or in front of the placenta; but, according to Taylor, the placenta never loses entirely its original relationship with the Fallopian tube.[2] Matthews Duncan taught the same.[3]

Bland-Sutton[4] points out that in some cases the dislocation of the placenta may be downwards till it is arrested on the pelvic floor, the embryo lying above the placenta, besides being dislocated upwards, as described above.

There is no doubt that these displacements of the placenta, besides being a constant source of danger to the mother, also imperil the life of the fœtus. An extra-uterine placenta is less perfect than a uterine one: it is an inefficient respiratory organ, and the placenta itself is constantly liable to damage. The result on the child is that an extra-uterine fœtus is often an 'unsatisfactory individual,' and is either ill-formed or defective in size.[5]

Berry Hart has studied extra-uterine placentæ, and finds that on the displacement or migration of the placenta from mucous to connective tissue (such migration being contrary to the views of Taylor) the placenta is gradually reduced to a mass of compressed villi, the serotina being replaced by blood crystals and organising blood clot, the shape of the placenta undergoing the most remarkable transformation.

Another point worthy of notice in the extra-uterine placenta is that, as previously stated, no true decidua serotina is formed, the tubal mucosa playing a very insignificant part, and the placenta is mainly, if not entirely, derived from fœtal tissues;[6] but in its growth 'it absorbs, takes up, and metamorphoses into its own tissue that of the Fallopian tube, ovary, and broad ligament, from which it receives its blood supply.' It is to be

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[1] Taylor, 'On some Cases of Ectopic Gestation,' *Brit. Gyn. Journ.* vol. viii. 1892, p. 168. (Note.—The adhesions of intestine and omentum, however, may prevent such 'exploration.'—Author.)

[2] Taylor, *Extra-uterine Pregnancy*, p. 80.

[3] Matthews Duncan, *Lancet*, vol. ii. 1889, p. 53.

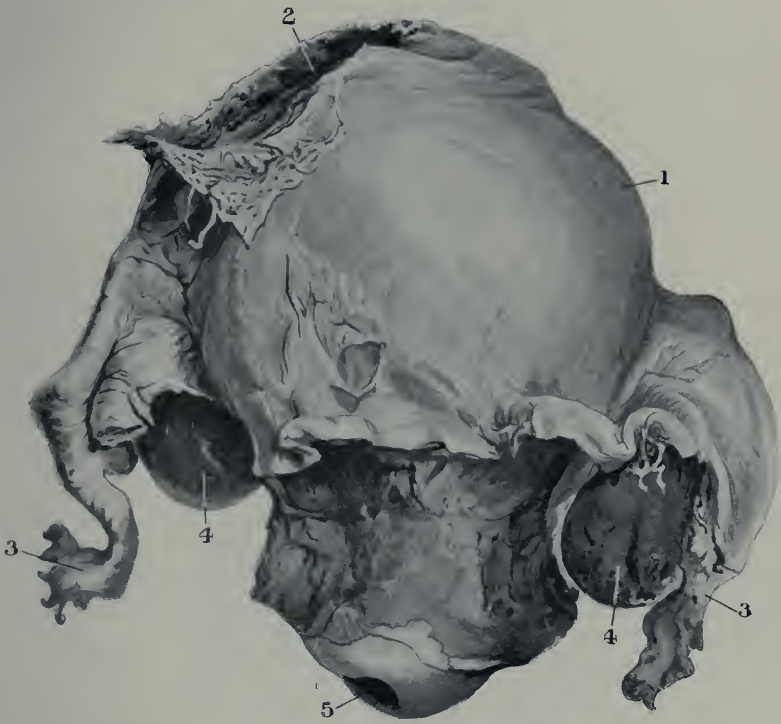
[4] Bland-Sutton, Allbutt and Playfair, *op. cit.* p. 465.

[5] *Id. op. cit.* p. 468.

[6] *Id. op. cit.* p. 464.

Fig. 54.

INTERSTITIAL (TUBO-UTERINE) PREGNANCY.



1. Fundus of uterus, above and to the left of which is a sac which contained the fœtus shown in fig. 55. The sac did not communicate with the uterine cavity. The uterus is 5 inches long and contained a decidua.
2. Site of the rupture which occurred into the peritoneal cavity at about the second month with fatal hæmorrhage.

3, 3. Fallopian tubes. Note the low attachment of the right-hand tube as compared with the left, and the asymmetry of the uterus, which is bulged upwards and to the left by the interstitial sac of the pregnancy. (Compare fig. 56, D.)

4, 4. Ovaries.

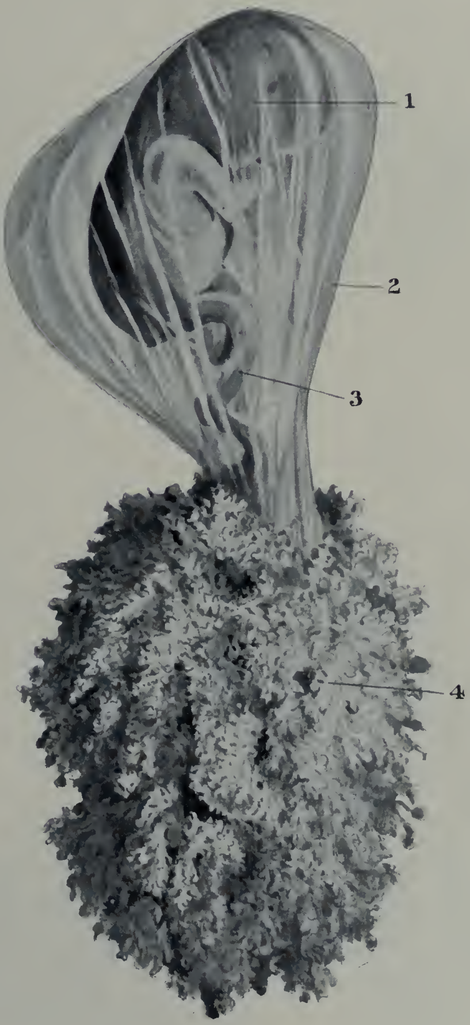
5. Cervix uteri.

(Specimen 4691. Museum, Royal College of Surgeons. Doran, 'Trans. Obstet. Soc. Lond.' vol. xxiv. 1882, p. 227.)

Fig. 55.

FŒTUS AND MEMBRANES FROM A CASE OF RUPTURED INTERSTITIAL (TUBO-UTERINE) PREGNANCY.

(The uterus is shown in fig. 54.)



1. Fœtus contained in the amnion, which has become detached and everted from the chorion. The fœtus is at about the second month of gestation.
2. Amnion.
3. Umbilical cord.
4. Chorion and placenta.

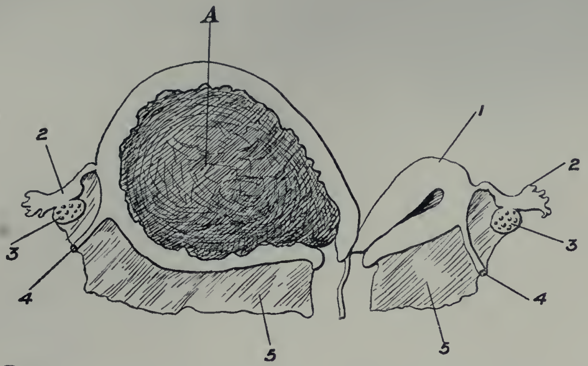
(Specimen 4691 A. Museum, Royal College of Surgeons.)

FIG.

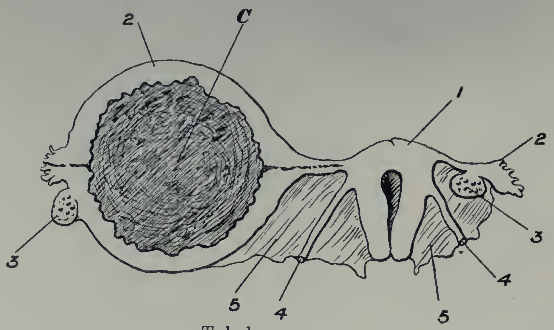
DIAGRAMS TO SHOW RELATIVE DIFFERENCES IN THE FOLLOWING CONDITIONS:

A. Pregnancy in one half of a double uterus;
B. Pregnancy in a rudimentary uterine horn;
C. Tubal pregnancy;
D. Interstitial (tubo-uterine) pregnancy.

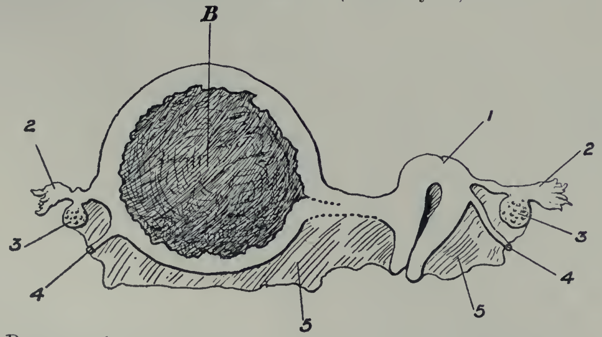
Note the relations of the sac to the round ligament in each.



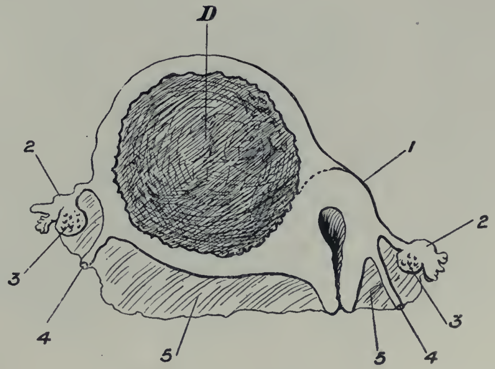
Pregnancy in one-half of a double uterus. The vagina is also represented as double. (After Taylor.)



Tubal pregnancy.



Pregnancy in a rudimentary uterine horn. There is supposed to be no connection between the rudimentary horn and the uterus. (After Taylor.)



Interstitial pregnancy (tubo-uterine). (After Taylor.)

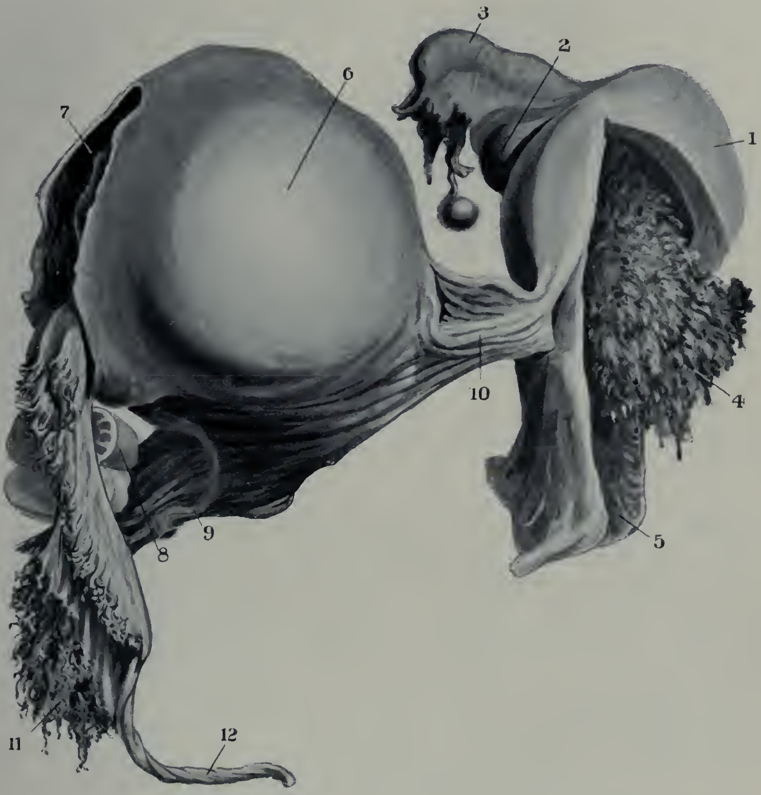
In each—1. Uterus. 2, 2. Fallopian tubes. 3, 3. Uterus. 4, 4. Round ligament. 5, 5. Broad ligament.

The pregnancy is represented by the darker-shaded areas.

(Taylor, 'Extra-Uterine Pregnancy,' fig. 54, p. 156; Roberts, 'Brit. Med. Journ.' vol. i. 1900, p. 1282.)

FIG. 57.

PREGNANCY IN A RUDIMENTARY HORN OF A UTERUS BICORNIS. RUPTURE AT THE FOURTH MONTH.



1. The well-developed half of the uterus containing a decidua.

2, 3. Ovary and tube belonging to the well-developed half of the uterus.

4. Decidua *in situ*.

5. Cervix of well-developed half of uterus.

6. Sac containing the pregnancy which has ruptured; it is connected (at 10) by a band of muscular tissue to the well-developed half of the uterus. This band has no channel within it. The sac of the pregnancy is really the imperfectly developed horn of a uterus bicornis.

7. Site of rupture.

8. Ovary belonging to the undeveloped half.

9. Round ligament outside the sac containing the pregnancy.

10. Band of union between the undeveloped half-uterus and the well-developed half. It is impervious.

11. Placenta and membranes.

12. Umbilical cord.

(Specimen 3078. Museum, St. Bartholomew's Hospital.)

noted that Taylor puts down defects in the child to pressure, rather than to the effects of malnutrition due to placental deficiency, and says that the children of ligamentary pregnancies may sometimes be free from deformity.[1]

Tubo-uterine or Interstitial Pregnancy. (Figs. 54, 55, 56 D.)—This is really an ectopic gestation, as it is outside the uterine cavity; it is a rare but most dangerous condition. The pregnancy grows in the portion of the tube traversing the uterine tissue near the uterine ostium. As it increases it raises the height of the uterus on the side affected, producing a curious and marked asymmetry and bulging of the cornu where the sac exists, so that the opposite tube looks as if it were at a much lower level (figs. 54, 55, 56 D). The pregnancy rarely continues to grow longer than the fourth month, and rupture is certain to take place into the abdomen, with very severe and diffuse hæmorrhage.[2] Rupture is said sometimes to occur into the uterus, but it is improbable.

It is possible, of course, for a tubal pregnancy eventually to invade the uterine wall, but this is not a true interstitial pregnancy.[3]

It is to be remembered, as pointed out by Virchow,[4] that the position of the round ligament is always on the uterine side of tubal structures, anything outside the point of its attachment being tubal in origin, anything on its inner side uterine. This is a useful guide in complicated cornual conditions and abnormalities (see fig. 56).

Cornual Pregnancy.[5] (Figs. 56 B, 57.)—Some cases of supposed pregnancy in the tube have been proved to be **pregnancy in a rudimentary horn of a uterus bicornis.**

The deformity is the result of failure of development of the Müllerian ducts; if both ducts coalesce but fail to communicate a uterus duplex or uterus bicornis is produced; if one duct only grows at a proper rate, the other remaining more or less stunted, a unicorn uterus is the result, the immature cornu being attached to the fully developed horn, near the upper part of the cervix, some distance below the fundus. The ovary on that side is often ill-developed. The round ligament can be traced to the rudimentary horn, outside of which is tube. A communication between the deformed horn and the well-developed one may or may not exist[1] (fig. 56B).

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[1] Taylor, *Extra-uterine Pregnancy*, p. 81.

[2] B. Hicks, *Guy's Hosp. Reports*, vol. vi. p. 275; Doran, *Trans. Obstet. Soc. Lond.* vol. xxiv. 1882, p. 227 (ruptured at second month), see figs. 54, 55; Bland-Sutton, *Surgical Diseases of Fallopian Tubes*, p. 352, new ed., p. 286.

[3] Taylor, *op. cit.* p. 85, and *id. Lancet*, vol. i. 1898, pp. 1447, 1515, 1668, 1739.

[4] Virchow, *Monat. für Geburtsh.* 1860.

[5] Kussmaul, *Malformations of the Uterus*, 1859; Turner, *Edin. Med. Journ.* 1866; Routh, *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 2; Handfield-Jones, *ibid.* p. 146.

In a well-formed uterus bicornis pregnancy may take place in either horn or both, if in one (fig. 57) a decidua forms in the other which is not impregnated, and both horns enlarge. In such cases gestation rarely causes trouble, and delivery may be natural; but if an impregnated ovum lodge in the rudimentary cornu of the unicorn uterus and it cannot pass into the other horn, rupture takes place, with disastrous consequences.[2] In these cases either transmigration of the ovum must have occurred, or the channel of communication between the rudimentary cornu and the cervical canal became occluded subsequent to impregnation.[3] The sac may rupture at any time between the second to ninth month,[4] or at full term an ineffectual labour leads to death and subsequent mummification of the foetus.[5] Axial rotation of the pregnant horn may occur, as in ovarian tumours.

Retention of the Fœtus.—Curious and remarkable changes follow the retention of a fœtus in extra-uterine pregnancy. The length of time that such fœtations may be retained varies. Over a dozen cases have been reported in which the pregnancy has been retained for more than twenty years, and in three or four, fifty years or longer.[6] In Leopold's[7] case it was retained thirty-five years, Cheston's fifty-two years,[8] Robert Barnes' forty-two years. Galabin[9] reports a case of intra-ligamentous gestation retained for twenty-one years; the tumour occupied the left broad ligament, and was dissected out of the cellular tissue; it was calcified, and on being sawn through a full-term fœtus in wonderful preservation was found. There was an ovarian tumour of the right side.

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[1] See very good drawing in Kelly, *Operative Gynæcology*, vol. ii. p. 465; see also specimen, St. Bartholomew's Hospital Museum, No. 3078. For literature see Bland-Sutton, *op. cit.* new ed. 1896, p. 295.

[2] St. Bartholomew's Hospital Museum, specimen 3078.

[3] Bland-Sutton, *Surgical Diseases of Ovaries and Fallopian Tubes*, p. 361.

[4] Fourth to fifth month (Taylor), p. 141.

[5] *Ibid.* p. 362.

[6] Taylor, *Extra-uterine Pregnancy*, p. 176.

[7] Leopold, *Arch. f. Gynäk.* vol. xix. pt. ii. p. 210, 1882; Bland-Sutton, Allbutt and Playfair, *op. cit.* p. 472.

[8] Cheston's case, quoted by Bland-Sutton, new ed., p. 282, in *Med.-Chir. Trans.* 1814, vol. v. p. 104.

[9] Galabin, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 38.

In retained fœtus the liquor amnii disappears; the placenta, owing to purely degenerative changes, grows more bulky for a few months after the death of the fœtus.[1] Afterwards the placenta is slowly absorbed, till no trace of it can be found; finally contraction of the sac takes place, lime salts are deposited around the fœtus, forming a hard calcareous shell (**lithopædion**), or the tissues of the fœtus undergo a fatty change or saponification, becoming converted into **adipocere**[2]; at last, only the skeleton remains.

Sometimes these retained, shrunken, and consolidated pregnancies are found in very curious situations in the abdomen.

Other troubles may arise from **decomposition**; pathogenic organisms attack the sac from some of the adjacent hollow viscera, and suppuration and toxæmia occur; the pus may find its way through the abdominal wall, or into the vagina, uterus, rectum, or bladder, and fragments of fœtal tissue escape.[3]

Owing to the near relationship of the rectum to the sac of late posterior tubo-ligamentary pregnancies, these are far more liable than any other variety to become septic or putrid.[4]

A case occurred at the Samaritan Free Hospital under Doran[5] where a putrid full-term fœtus was removed from a posterior intra-ligamentary pregnancy a week after the spurious labour at term. The author assisted Meredith at an operation on a similar case at the Hospital in 1893, in which, after removal of the putrid fœtus, several fæcal fistulous tracks were found communicating with the sac.[6]

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[1] Knowsley Thornton, *Trans. Obstet. Soc. Lond.* vol. xxiv. 1882, pp. 85, 86; and specimen Mus. R.C.S., Pathol. Ser., No. 4698.

[2] Bland-Sutton, *Surgical Diseases of Ovaries and Fallopian Tubes*, p. 281.

[3] M. Shield, *Trans. Obstet. Soc. Lond.* vol. xxxiii. 1891, p. 148; and Gervis, *Med.-Chir. Trans.* vol. lxx. p. 35, quoted by Bland-Sutton, new ed., p. 284.

[4] Taylor, *Extra-uterine Pregnancy*, p. 179.

[5] Doran, *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, pp. 8 and 213; see also J. Phillips, *ibid.* p. 121.

[6] Freund noted that the diarrhœa sometimes observed in the later stages of extra-uterine pregnancy continued to term was not so much a result of sepsis as a sign that the placenta was partly attached to intestine which became abnormally vascular, so that catarrh occurred (*Edin. Med. Journ.* vol. xxix. 1883–4, p. 526). Intestinal attachments greatly endanger the fœtus.

CHAPTER XII

ON UTERINE FIBROIDS

Uterine Fibroids, also known as myomata, fibromata, or fibro-myomata, are by far the most common of uterine new growths. How common they are can only be answered approximately, and the question rests upon both post-mortem and clinical experience. Clinical records may understate their frequency, or may fail to discover them, since they may not necessarily enlarge the uterus to any great extent, or give rise to any symptoms whatever.

With regard to post-mortems, at all events on hospital patients, difficulties arise, for either the patient may die of something else, and the presence of small fibroids may be overlooked, or permission for a post-mortem cannot be obtained. In order to answer the question approximately we should require to know the results of post-mortem examination of all female members of the community. It is impossible to answer the question numerically, 'What percentage of women have fibroids?' It is quite certain they are very common, and this is generally admitted.

The author would refer the reader to Champneys'[1] address 'On some Points in the Natural History of Uterine Fibroids' for further discussion on this point and others.

Haultain[2] gives Bayle's[3] data that fibroids occur in 20 per cent. of all women over thirty-five years of age; in women of fifty, Klob estimates their occurrence at 40 per cent.[4]

Champneys, taking the post-mortem records at St. Bartholomew's Hospital for over thirty years, finds that 1860 necropsies were made on women over thirty years of age, and that in 141 instances (8 per cent.), quite apart from the cause of death, fibroids were found of greater or smaller size; out of these 141 cases, 3 deaths were attributable to the fibroid apart from operation, *i.e.* a death-rate of about 2 per cent.

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[1] Champneys, *Lancet*, vol. i. 1900, p. 147, finds that the frequency of fibroids is ·01 per cent. in a population of 959,217; see also discussion on 'Natural History of Fibroids,' Brit. Med. Association, *Brit. Med. Journ.* vol. ii. 1900, p. 713.

[2] Allbutt and Playfair, *op. cit.* p. 562.

[3] See also Roger Williams, *Brit. Med. Journ.* vol. ii. 1900, p. 718.

[4] Herman, *op. cit.* p. 406; and Klob, *Wiener Med. Wochenschrift*, 1863.

At the Edinburgh Royal Infirmary, out of 2230 gynæcological cases only 8 per cent. were diagnosed as fibroids (Haultain).

Herman[1] says that in his out-patient records fibroids were diagnosed in about 3 per cent. of the cases, and taking the out-patients, 40 per cent. were over thirty-five, so that the proportion of women over thirty-five seeking advice for fibroids would be about 7½ per cent, whilst the percentage of fibroids in women over thirty-five ascertained by post-mortem examination is, as shown above, much higher. This is very important, because of late some have urged that every fibroid should be removed as soon as it is discovered, either to relieve existing symptoms or to prevent trouble in the remote future.

Fibroids are not to be removed merely because they exist, as the mortality apart from operation is only 3 in 2,160,112 cases, or ·000138 per cent.[2] It seems undeniable that deaths from fibroids apart from operation are excessively rare. Operation is only necessary for the few cases where the symptoms caused by the fibroid are undoubtedly injuring health, and where, from the situation of the tumour, it is almost certain to become a source of danger (Herman).

The above statements are perhaps not essentially pathological in their significance, but the author makes no apology for bringing them forward in the present work.

Etiology.—Nothing is known as to the cause of fibroids: they occur chiefly between the age of thirty-five and forty-five. They are very rare before twenty-five, or at least rarely diagnosed. Spencer,[3] however, gives a record of two cases of his own occurring under twenty-five, with records of forty others. There is no satisfactory record of any case before the age of puberty.

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[1] Herman, *Diseases of Women*, p. 407.

[2] Champneys, *Lancet*, vol. i. 1900, p. 149. (Note.—These views have been criticised, and although no doubt the mortality from fibroid tumours is very low, 'yet Dr. Champneys' method of determining the percentage mortality by dividing the number of fatal cases seen in the post-mortem room into the number of female out-patients during a given period appears liable to fallacy.'—*Lancet*, vol. ii. 1900, p. 1887.)

[3] H. R. Spencer, 'Two Cases of Fibro-myoma removed from women under twenty-five,' *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 228. (Note.—A case occurred in the Samaritan Free Hospital, Feb. 1901, in the author's ward, under the care of Lockyer, in a woman of twenty-three: it was removed by enucleation. The diagnosis of pregnancy was made before operation. She made a good recovery.)

From what existing structures can fibroids arise? The uterus is a muscular organ invested with peritoneum directly continuous with the folds of the broad ligament; the thickness of its walls consist of unstriped muscle which is really a muscularis mucosæ.[1] The true sub-serous investment is a layer of unstriped muscular tissue, directly continuous with the uterine muscle and that of the broad ligament belonging really to the peritoneum. The endometrium itself also contains some muscular fibre. These three coats, the muscular wall, the sub-serous coat, and the endometrium, all play their part in the origin of fibroids.

The sub-serous peritoneal coat cannot be separated in the adult from the muscular, but in childhood they are more distinct.

Other structures containing unstriped muscle are the round ligament, the broad ligament, and ovarian ligament. The unstriped fibres in the broad ligament (the uterine platysma) are often well seen in cases of a burrowing broad ligament cyst, stretching over the tumour in strands, which contract and wrinkle, and which diminish the capsule after enucleation of the cyst.

From this standpoint we may attempt a classification, at all events as regards **origin**, into—

a. **Fibroids of the Body of the Uterus.**
    1. Intramural fibroids, arising from true uterine tissue.
    2. Sub-mucous, from mucous membrane.
    3. Sub-peritoneal, from the sub-serous or peritoneal coats of the uterus, the broad ligament, round ligament, or ligament of the ovary.

b. **Fibroids of the Cervix.**
    1. Intra-cervical.
    2. Sub-serous cervical.

Fibroids of the Body of the Uterus.—It is stated that all fibroids arise in the muscular wall of the uterus, and from this grow either inwards or outwards. It may be so in a large number of cases, but it does not explain all, especially curious broad ligament fibroids of which more will be said later.

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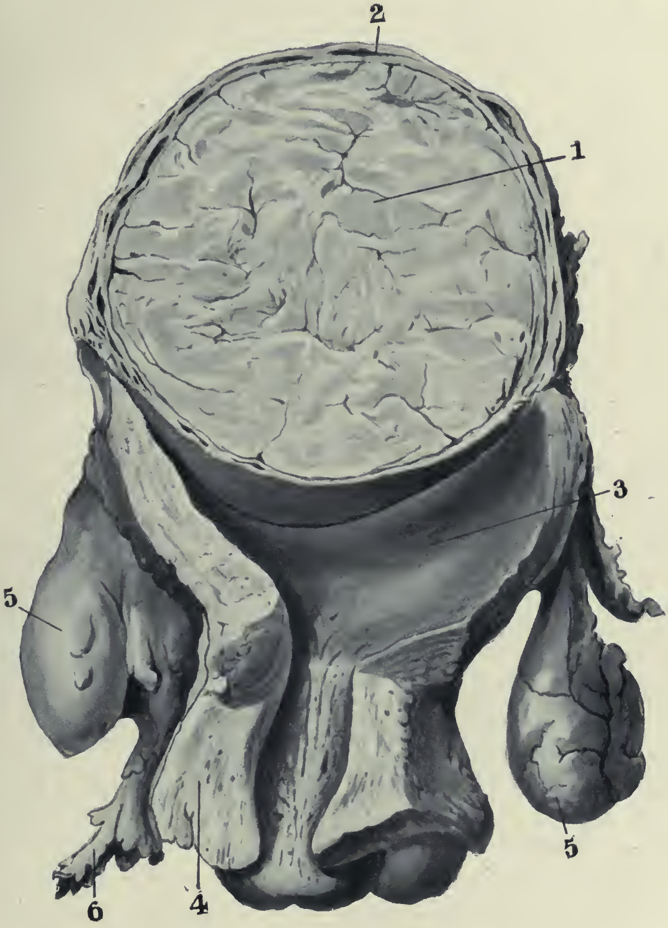
[1] Sir J. Williams, 'On the Circulation in the Uterus, with some of its Anatomical and Pathological Bearings,' *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, pp. 112, 114.

Fig. 58.

UTERINE FIBROID (TO SHOW CAPSULE AND THE ORDINARY STRUCTURE OF SUCH TUMOURS).



An intra-mural fibroid of the fundus uteri. The ordinary appearance of a fibroid on section is shown. The capsule is very well marked, and exhibits many large venous sinuses. The patient died of hæmorrhage due to the spontaneous opening of a uterine sinus at the lower part of the tumour.

1. Main mass of the fibroid.
2. Capsule showing a large venous sinus.
3. Cavity of uterus.
4. Cervix.

5, 5. Ovaries, which are enlarged and œdematous.

6. Fallopian tube.

(Specimen 2985. Museum, St. Bartholomew's Hospital.)

Fibroids may also be classified as hard and soft, according to their structure, and rate of growth.

Hard Fibroids are the commoner: they are less vascular and have the typical white appearance on section after removal, and are generally encapsuled (fig. 58): they contain more fibrous than muscular tissue, and their growth is generally slow. The tumour itself is made up of a disorderly interlacement of muscular and connective tissue, the fibres in the larger masses being grouped into more or less well-defined whorls or nodules. Between the groups of fibres run arteries, veins (and probably lymph channels), derived from the normal vessels of the uterus, ramifying at first beneath the capsule of the tumour, from which, if it exists, the main blood supply of the tumour is derived, and which contains large venous sinuses.

The vessels from the capsule plunge into the fibroid vertically, mostly running in the trabeculæ of connective tissue which separate the various masses of the tumour from one another; eventually the vessels become less well marked, but some arterioles appear to end in the centres of the whorls of concentric fibres and to be terminal. The harder the fibroid the less vascular it is; in fact, by its very growth a fibroid tends to deprive itself of the blood it wants for its nutrition, and this may reach such a stage that deeply placed areas of the fibroid begin to decay and undergo degenerative changes, while the peripheral parts of the tumour being still well nourished from the capsule are healthy. It is suggestive, considering these changes, that each central arteriole[1] is the starting point of the fibroid changes around it, and that in some such manner fibroids start, probably as a proliferation, and that such proliferation eventually reaches a stage when, by pressure, adequate nutrition is no longer possible, and decay and necrosis begin. Necrosis, at all events in the majority of cases, commences in the centre of the fibroid. Hard fibroids generally cease to grow after the menopause.

Soft Fibroids are less common than the hard type, but a good deal depends upon what is exactly meant by 'soft.' Some fibroids are soft and rapidly growing, very vascular, and more or less part of a general uterine enlargement, with practically no capsule, the mass being single, and not in many nodules as in the hard variety. Such fibroids may continue to grow after the menopause, or may at any time take on a rapid growth, and are little influenced by ordinary treatment or by the removal of the ovaries. They are said to contain more muscular tissue; but this is not always the case, though some certainly appear redder, and in fact look like liver on section.

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[1] Kleinwächter, 'Zur Entwickelung der Fibro-Myome des Uterus,' *Zeitschrift für Geburtsh. u. Gynäk.* vol. ix. 1887, p. 68, and *Id.* 'Zur Biologie der Fibro-Myome des Uterus,' *ib.* vol. xxv. 1893, p. 2; and Bland-Sutton, *Lancet*, vol. i. 1901, p. 812.

Other fibroids are soft, and even rapidly growing, from œdema, or are breaking down in the centre.

The distinction of fibroids into **intramural, subperitoneal** or **sub-mucous** is sometimes attempted, but such a sharp definition is often impossible. The nearer a fibroid approaches in its growth to the surface the more will it project either towards the peritoneal surface or towards the uterine cavity. Sometimes a fibroid is wholly sub-peritoneal, and is either sessile or pedunculated; multiple sub-peritoneal growths are very common, and one or more of them may attain a prodigious size.

Intramural Fibroids may be single or multiple: they generally have distinct capsules, and are usually of the hard variety; others, as before mentioned, may have no capsule to speak of, and are exceedingly vascular, so that on section they look not unlike cavernous nævi (Myoma Telangiectodes).[1] Such tumours during life furnish a loud venous hum on auscultation.[2]

Sub-mucous Fibroids.—These probably arise in the deeper layers of the mucous membrane, or an intramural tumour may grow towards the uterine cavity. Such sub-mucous fibroids may remain sessile, or become stalked, and are termed 'fibroid polypi.' Sub-mucous fibroids are covered by the mucous lining of the uterus (columnar ciliated epithelium), but when the polypus enters the vagina the surface exposed to pressure frequently becomes covered with stratified squamous cells. This may also occur on the surface of a polypus while still contained within the uterus.

Fibroids of the Cervix.[3]—Bland-Sutton has drawn attention to this class of fibroids, which seem to have special features of their own.

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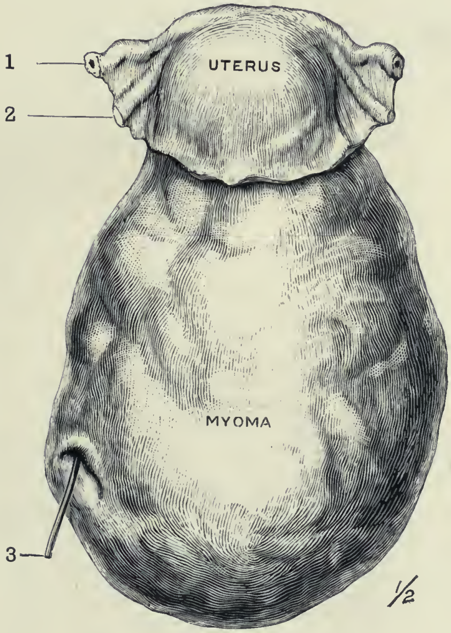
[1] Virchow, *Geschwulstlehre*, iii. p. 195; also Haultain, 'Benign Growths of the Uterus,' Allbutt and Playfair, *op. cit.* p. 591.

[2] Bland-Sutton and Giles, *Diseases of Women*, p. 184.

[3] Bland-Sutton, *Clin. Journ.* Feb. 9 to Mar. 28, 1898; W. Tate, 'Calcareous Fibroid of Cervix,' *Trans. Obstet. Soc. Lond.* vol. xli. 1889, p. 372.

Fig. 59.

MYOMA OF THE NECK OF THE UTERUS.



An intra-cervical myoma; it is oval in shape, and the uterus is perched on its summit. The tumour weighed 5 kilogrammes. The fundus of the uterus was on a level with the navel.

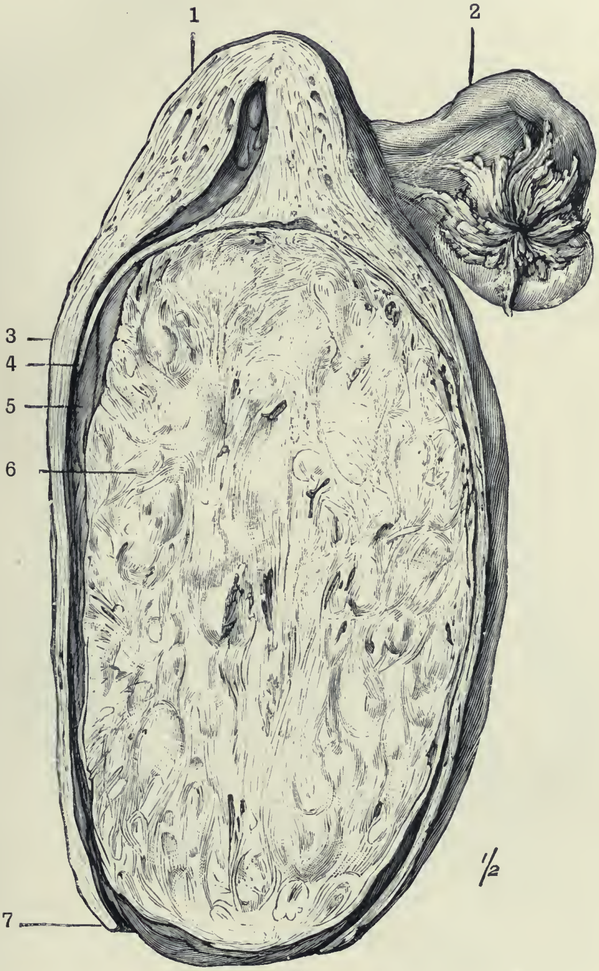
1. Fallopian tube. 2. Round ligament. 3. External os.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxix. p. 161.)

Note.—Myoma, fibro-myoma, and fibroid are synonymous terms.

FIG. 60.

MYOMA OF THE NECK OF THE UTERUS.



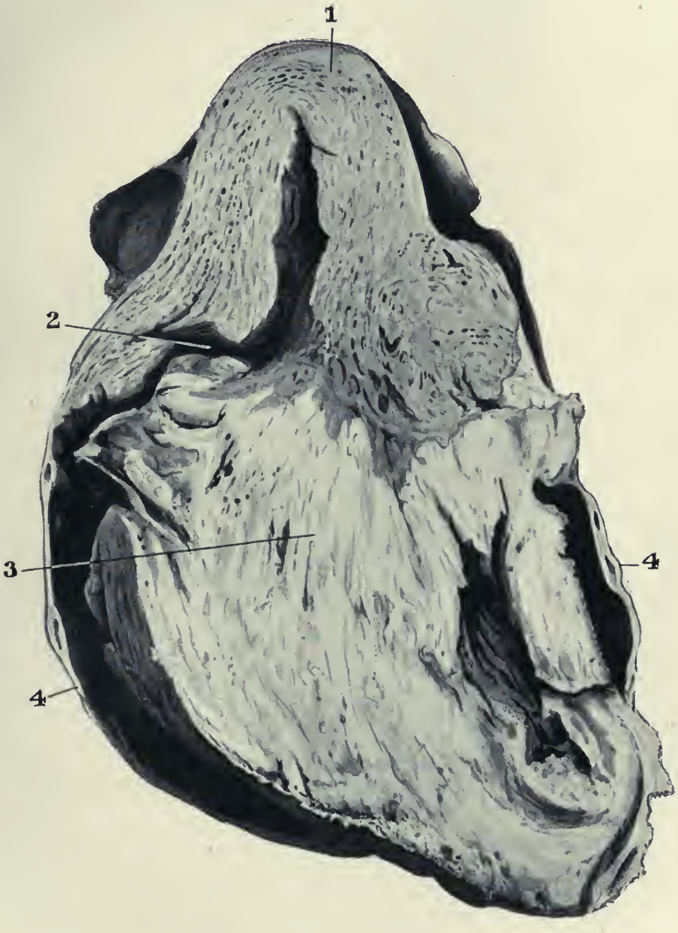
An intra-cervical myoma in sagittal section, showing the characteristic elliptical outline; the expanded walls of the cervical canal extend like a thin capsule around the tumour. The cervical canal runs along the periphery of the tumour. Structurally it exhibits the characteristic whorled appearance of uterine fibroids, and is microscopically identical with them.

1. Uterus.
2. Tube.
3. Cervix.
4. Cervical canal.
5. Mucous membrane.
6. Myoma.
7. External os.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxix. p. 162.)

Fig. 61.

LARGE FIBROID OF THE CERVIX.



A vertical antero-posterior section of a uterus with a large fibrous growth arising from the posterior wall of the cervix. No definite capsule exists, and the surface of the tumour occupying the vagina is ulcerated and necrosed. A large cavity due to degeneration occupies its lower and right-hand portion. The uterus is very vascular near the attachment of the tumour.

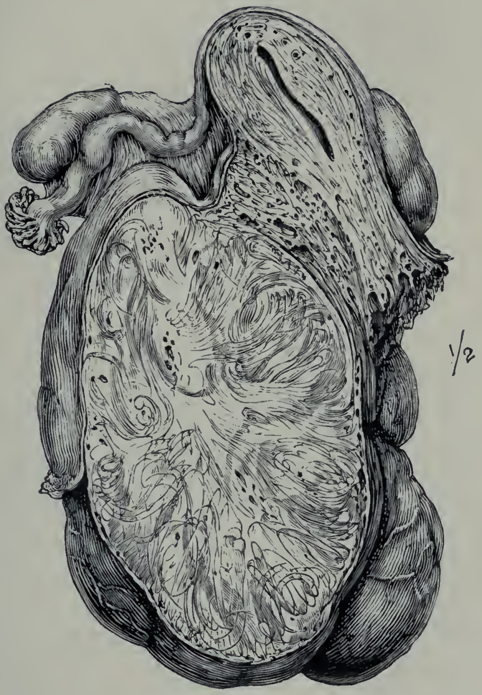
1. Fundus uteri.
2. Much-distorted canal of cervix.
3. Main mass of the tumour.

4, 4. Vagina, into which most of the tumour has been expelled; part of it protruded through the vulva.

(Specimen 4617. Museum, Royal College of Surgeons.)

Fig. 62.

MYOMA OF THE NECK OF THE UTERUS.



A subserous cervical myoma in sagittal section, showing its elliptical outline. The tumour grows from the outer aspect of the cervix, but does not invade the cervical canal. The body of the uterus was pushed high above the symphysis. The ovoid shape is probably due to moulding by the hard parts of the true pelvis. Such tumours may give rise to serious pressure symptoms, the urethra or ureters being often affected.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxix. 1897, p. 163.)

Fibroids of the cervix probably arise in the tissues of the cervix itself, and do not come down from the body of the uterus as was supposed: they may grow to a considerable size and give rise to most severe symptoms. Two classes of cases are met with:

a. **Intra-cervical.** (Figs. 59, 60, 61.)—Such a tumour arises in the tissues of the cervix, generally to one side or the other, with a characteristic elliptic outline from above downwards, and having a shell of the cervix around it like a capsule, while the body of the uterus lies above this like a cap. There is often great distortion of the cervical canal (figs. 60, 61). The elliptic shape of such cervical fibroids is probably determined by the osseous boundaries of the true pelvis, and in most cases of large tumours of this sort the pressure on pelvic viscera or on the ureters or urethra is most injurious; in fact, pressure symptoms are the chief characteristics in cervical fibroids.

b. **Sub-serous Cervical Fibroids.**—This second variety (fig. 62) also grows from the cervix, but instead of merely distending the cervix it grows outwards beneath the peritoneum, generally to one side, into the broad ligament, producing great distortion and lifting the uterus itself high up into the abdominal cavity out of the pelvis. Some of these tumours are of considerable size, and the body of the uterus may reach the umbilicus.[1]

The interest of these cases is chiefly their dangerous pressure symptoms, and their involvement of the broad ligament. They must be distinguished from true broad ligament fibroids. Careful dissection shows their connection with the cervix.

Conditions of the Uterus and Appendages with Fibroids.—In nearly all cases the uterus itself is enlarged, not rarely to a very great degree, and may be monstrously misshapen. Sometimes the enlargement is rounded and smooth, without distinct lumps on its surface. In other cases the abdomen is occupied by a huge mass of nodular irregular tumours of all sizes, some large, some small, some sessile, some more or less pedunculated, all arising from an enlarged mass in the pelvis, which may be proved to be the distorted uterus, and from which other nodular outgrowths may grow outwards into the broad ligament, or down into Douglas's pouch. Dissection of such a uterus may discover everywhere in its walls bosses of all sizes, themselves fibroids; in other places fibroids are seen involving the cavity of the uterus, becoming sub-mucous or even polypoid.

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[1] Bland-Sutton, 'Cervical Fibroid weighing nearly seven pounds,' case i., *Brit. Med. Journ.* vol. ii. 1896, p. 1308; see also *id. Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 298.

Most of the pedunculated sub-peritoneal tumours are very hard, and are more or less destitute of capsule, though they are covered with altered peritoneum. The length of the pedicle varies. Some fibroids of this nature are extremely mobile, and their occurrence in the abdomen in places far removed from the pelvis often leads to a wrong diagnosis. Sometimes the shape of these tumours imitates other viscera, and large flat cake-like notched fibroids have been mistaken for the spleen.

The stony hardness of some of these sub-peritoneal tumours is remarkable, and they are frequently calcified. Sometimes large single fibroids are cystic, and distinct fluctuation can be obtained; others, though very soft from internal degeneration and œdema, do not fluctuate. Again, others are soft from extreme vascularity. There is no distinct rule as to the enlargement of the uterus by fibroids; all forms of fibroids may exist at the one time; but practically the two types met with are either a smooth general enlargement or a nodular bossed tumour or tumours.

In fibroids that involve the pelvis, or where the tumour is lifted up above the brim, it may be quite impossible to determine the **position of the cervix** at all by vaginal examination.

The distortion and great **lengthening of the canal of the uterus** on passing the sound is often remarkable.

With large fibroids certain changes are sometimes found in the tubes, ovaries, and broad ligaments. Another great point is the enormous increase both in the size and complexity of the **vessels**[1] which supply the tumour and in the venous plexuses of the broad ligaments. Sometimes there are huge œdematous areas in the same situation: they have been called by some dilated lymphatics, but the author takes them rather to be the results of backward pressure and œdema; anyhow these spaces contain serous fluid, and are beneath the peritoneum.

The **Fallopian tubes** are often enormously lengthened, but there does not appear to be much tendency to increase in thickness. It is not uncommon to find a certain amount of hydro-salpinx, or even hæmato-salpinx, with fibroids, the fimbriæ being glued together by local peritonitis; at other times the fimbriæ are natural, but very vascular and luxuriant.

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[1] In some cases the distribution of the vessels supplying large tumours is very irregular, and the greatest difficulty may be met with in securing them by ligature, in removing such growths.—Author.

The **ovaries** with fibroids are liable to many changes: sometimes they are small, deeply crenate, and very hard; in others large, elongated, smooth, flabby bodies with a tough capsule are found which are highly œdematous. These ovaries practically contain no corpora lutea, and the stroma is very myxomatous. The vessels are enlarged and thickened, and appear to have undergone some obliterative changes (endarteritis).

The great distortion in the position of the appendages by fibroids is remarkable: sometimes they can only be found at operation with the greatest difficulty, and cannot be removed with safety without the uterus.

The **round ligament** is often very much enlarged and may contain a large vessel or vessels.

Any local peritonitis affecting a fibroid leads to great changes in various structures, and possibly adhesion; and on the surface of some fibroids curious milky plaques of thickened peritoneum are found as the result of inflammation. Hydro-peritoneum is very rare. Sometimes the most remarkable changes are found in the **endometrium**. The whole endometrium is involved in an overgrowth of its elements, wrongly called 'endometritis,' but it is not as such an 'itis' at all. The condition is really adenoma of the body of the uterus; vessels, glands, and stroma share in the general overgrowth, and the mucosa may sometimes be ½ in. thick, or pedunculated adenomata (mucous polypi) are found.[1] This increase in thickness is not so in every case, but appears to occur mostly in uteri with large sub-mucous fibroids; with sub-peritoneal growths the endometrium may not be affected at all or may even be atrophied. Atrophy of the mucosa in uteri containing fibroids which project into its cavity is also seen. On the surface of such a tumour there is no endometrium, or only a few atrophied remains; in the depression into which such a tumour fits in the opposite wall a similar atrophy of the endometrium is found: it is due to pressure; while around the tumour is a distinct collar or corona of very vascular endometrium, and at its very edge many of the delicate vessels of the endometrium are ruptured. Exactly the same condition occurs in the opposite pit into which the fibroid fits: deep in the hollow no endometrium, at the edges of the cup[1] thick endometrium and a vascular corona of ruptured vessels. The rest of the mucosa of the uterus is as a rule uniformly thick and vascular.

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[1] See chapter on 'Endometritis.'

These changes are important in relation to the bleeding of fibroids, for the hæmorrhage comes from the endometrium, and not from the tumour. We do not know much even about menstruation, still less of the exact changes of the endometrium in relation to the menorrhagia of fibroids. Not all fibroids cause bleeding, but only those that involve the uterine cavity; and even then the hæmorrhage may be due to a local endometritic condition, mucous polypi, or even cancer of the endometrium,[2] and not to the fibroid itself.

The **catarrh** and profuse watery mucoid discharge coexisting with some fibroids, especially fibroid polypi, is probably due to the great increase of the glandular elements of the mucosa. This is interesting in the study of the normal discharges from the body of the uterus.

True endometritis may affect a uterus with fibroids, but here it is an infective process, the fibroid itself being in a state of necrosis and sloughing: it is a very dangerous condition (fig. 67).

Changes in Fibroids with Menstruation.[3]—There is monthly enlargement and monthly shrinking, together with an exaggeration of the normal menstrual phenomena. The uterus reaches its largest size just before the period begins, the flow commences, and the uterus rapidly shrinks.

Uterine contractions can even be felt in large tumours. Patients are sometimes aware of this increase in size, and if a tumour just fits the pelvis, pressure symptoms only occur just before the monthly flow, the period comes on, and they are comfortable. It is a sort of safety-valve action.

An instance of uterine contractions is found in the so-called 'intermittent fibroid polypus' which only appears at the cervix during menstruation, for during menstruation the polypus comes down, afterwards retreating as the cervix closes.

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[1] Howard Kelly, *Operative Gynæcology*, plate 19, vol. ii. p. 338.

[2] For 'Cancer of the Endometrium associated with Fibroids' see Cullen, *Cancer of the Uterus*, p. 439 *et seq.*

[3] Sir J. Williams, 'On some Periodic Changes which occur in Fibroids,' *Lancet*, vol. i. 1880, pp. 764, 873; see also Champneys, *Lancet*, vol. i. 1900, pp. 147, 174.

Growth of Fibroids.—Nothing is known of the growth of fibroids, either when they begin or when they cease, or why they remain stationary for a time, and then grow rapidly. Some of these sudden increases are due to œdema. As a rule fibroids grow slowly; the size of a fœtal head will rarely be reached under a year, that of a man's head under three years, and that of a full-term pregnancy under twelve years. How different this from an ovarian dropsy, which will grow to the largest size in the course of two or three years, and be fatal! [1]

It is generally believed that fibroids cease to grow at the menopause, which they seem to delay, but some may grow after it (Kleinwächter).

Some fibroids grow slowly and never become large, others are enormous. Richter [2] quotes a case where a 'fibroid' in a woman of twenty-three grew in two and a half years so large that the girth of the belly was 65 inches. A fibroid has been observed weighing 140 pounds.[3]

Absorption of Fibroids.[4]—Cases of this kind are on record, but in most of them the diagnosis was probably wrong, others undoubted, where the fibroid was of considerable size. Nothing is known of the pathology, and it is very rare.

Doran [5] has reported such a case, adding records of thirty-seven others with the literature. Other methods by which a so-called natural case of fibroids is brought about are expulsion, disintegration, and gangrene.

Pathological Changes in Fibroids.—These are of extreme interest, and include œdema, various degenerative changes, inflammation, suppuration and gangrene, calcification, and cystic and malignant degeneration.

Fibroids are most prone to degeneration; in fact, the larger a tumour is, the more likely is it to undergo some degenerative process. Most of them outgrow as it were their power of nutrition, and degeneration is the consequence.

A common condition met with is **œdema.** In such a tumour when cut open, instead of dense healthy fibrous tissue everywhere compact, one spot, if not many, in the tumour is undergoing a liquefactive change. Spaces are seen to exist filled with yellow fluid serum, which soon drains away from the section, and considerable diminution of the bulk of the tumour occurs; the serum itself sometimes coagulates spontaneously and is highly albuminous. Section of the fibrous tissue of such tumours displays the fibres swollen and disintegrated and their nutrition at a low ebb, and the vessels few and thickened. Sometimes such a tumour is like a honeycomb on section, everywhere occupied with cystic spaces, or there may be one larger irregular cavity crossed in all directions by trabeculæ of fibrous tissue, all that remains of the original tumour. Most of these tumours have a distinct capsule.

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[1] Matthews Duncan, *Clin. Lect.* p. 329, 4th ed.

[2] Herman, *Diseases of Women*, p. 413.

[3] Clarence Webster, *Diseases of Women*, p. 470.

[4] Schroeder, *Lehrbuch*, p. 230.

[5] Doran, 'On Absorption of Fibroid Tumours of the Uterus, with report of a suspected case,' *Trans. Obstet. Soc. Lond.* vol. xxxv. 1893, p. 250.

Some of these tumours grow to an immense size,[1] and the whole tumour is distinctly **cystic** (fig. 63) with more or less thin walls, or the walls are thinned in certain areas (fig. 64), in others only part of the tumour is affected. The question of cystic fibroids is important. Leopold and Fehling have described cavities in such tumours lined with a definite endothelium, and they think that the spaces are lymphatic in origin. The author has examined many ‘cystic’ fibroids of the body of the uterus for endothelium, but has never been able to demonstrate its occurrence. Cullingworth[2] also points out that these spaces are not true cysts. A curious fibro-adenomatous tumour growing in the cervix full of cysts lined with epithelium is in St. Bartholomew’s Hospital Museum[3] (fig. 65).

From mere œdema such cases may also pass through myxomatous[4] changes, and so low is the vitality of disintegrated fibroids that they are liable at any time to septic infection, and may be converted into mere abscess cavities[5] or break down and become gangrenous. Œdematous fibroids sometimes grow very rapidly and may as rapidly decrease. Œdema may affect cervical fibroids as well as fibroids of the body of the uterus.

Hæmorrhage into Fibroids.—Sometimes a fibroid appears on section distinctly red or liver-coloured; such tumours are generally single and encapsuled: they are thought by some to be nearly all muscular, on account of their colour. The author

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[1] H. Kelly, *Operative Gynæcology*, vol. ii. p. 343, quotes a case weighing 195 lbs.

[2] Cullingworth, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 8.

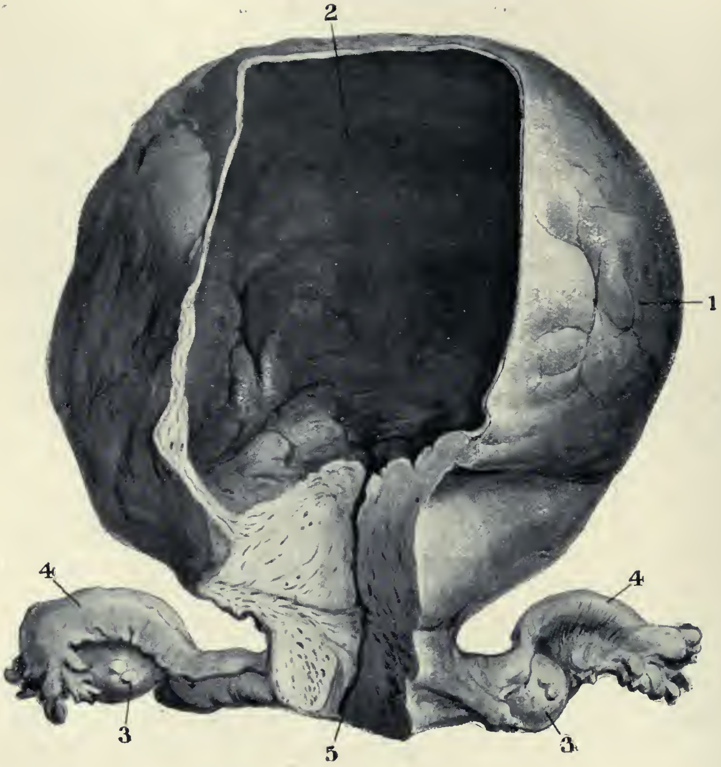
[3] Specimen 2960 B.

[4] Gusserow, *Neubildung des Uterus.*

[5] Suppurating Fibroid, 4607 B, Museum Royal Coll. Surg., Path. Ser.; and Roberts, *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 295.

FIG. 63.

CYSTIC FIBRO-MYOMA.



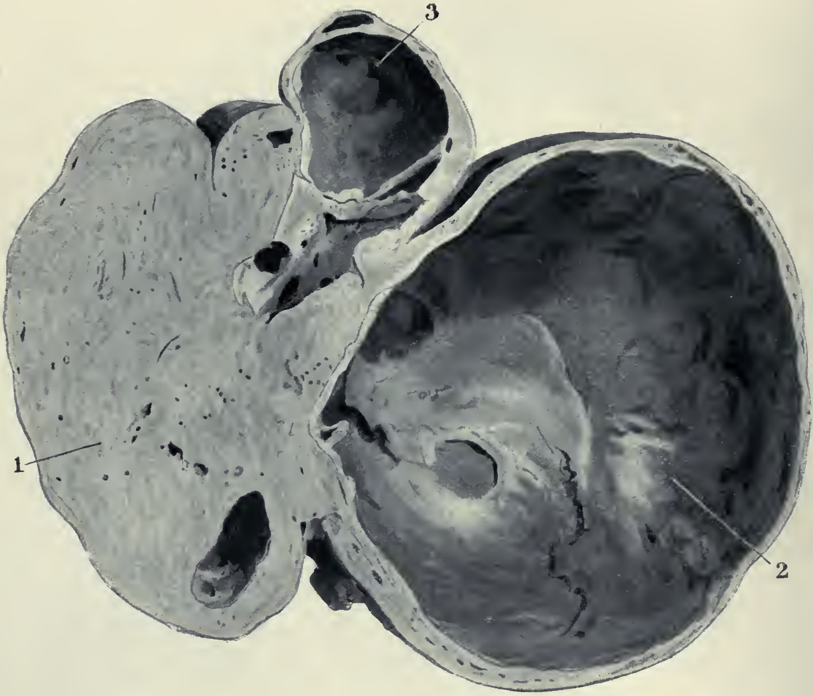
Portion of a uterus and corresponding ovaries and tubes seen from the front. A large unilocular cystic tumour is attached to the fundus. The tumour is oval in outline, and measures 8 inches by 5 inches; its wall consists of a thin layer of unstriped muscle and peritoneum. There is no lining membrane to the cavity, its interior being covered with shreds of blood-clot. Histologically it is a cystic fibro-myoma. It was removed from a single woman of 43.

1. Wall of fibroid, showing great thinning.	4, 4. Fallopian tubes.
2. Cavity of the tumour.	
3, 3. Ovaries.	5. Cavity of uterus.

(Specimen 4643 D. Museum, Royal College of Surgeons.)

Fig. 64.

CYSTIC DEGENERATION OF A UTERINE FIBROID.



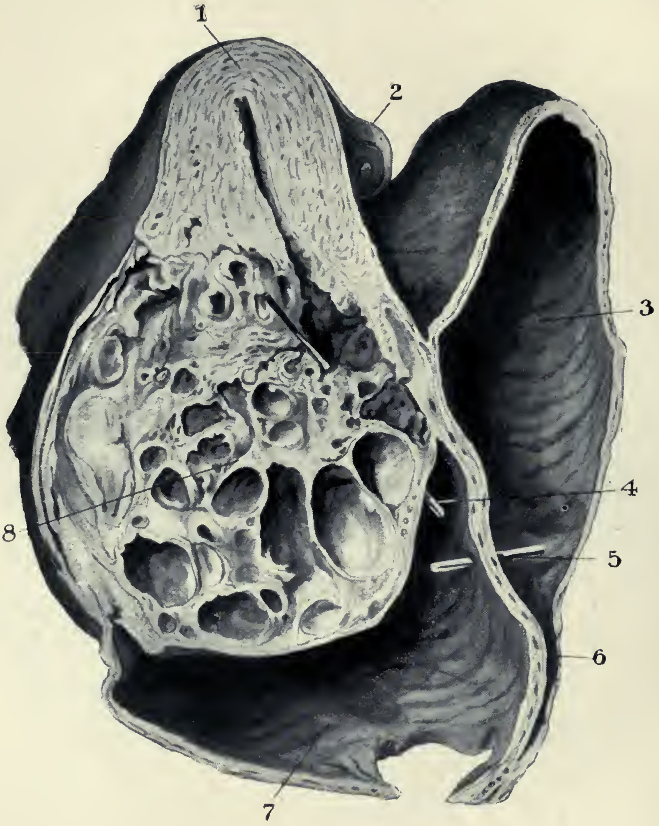
Portion of a large subperitoneal fibro-myoma which sprung from the fundus uteri. It has undergone extensive cystic degeneration. Removed by operation from a single woman of 29.

1. Solid portion of the tumour, showing a few cystic spaces.
2. Large cystic cavity, measuring 7 inches in diameter. It has no distinct lining membrane. Its outer surface is covered with peritoneum.
3. Another cystic cavity of a similar nature.

(Specimen 4643 c. Museum, Royal College of Surgeons. The other half of the specimen is in the Museum of the Middlesex Hospital.)

Fig. 65.

CYSTIC FIBRO-ADENOMA OF THE CERVIX.



A uterus in section, showing a large tumour occupying the cervix and protruding into the vagina. It is everywhere riddled with cysts. Microscopical examination shows the tumour to be a fibro-adenoma. Each cyst is lined with a delicate layer of columnar epithelium resembling that of the glands of the cervix.

1. Fundus uteri.
2. Ovary and tube.
3. Bladder.
4. Glass rod in canal of cervix.
5. Glass rod in small vesico-vaginal fistula.
6. Urethra.
7. Vagina.
8. Cystic tumour growing in the lower portion of the cervix, which forms its capsule.

(Specimen 2960 b. Museum, St. Bartholomew's Hospital.)

has examined a few of these cases, and has come to the conclusion that the condition represents degeneration and blood stasis; probably it is a sort of thrombosis, much the same as in an ovarian cyst with a twisted pedicle. The muscular fibres are poorly nourished and have lost their outlines, and the whole tumour is engorged with blood. The changes are degenerative, not those of active growth.

Vascular Fibroids.—A few cases of this nature are on record: they are very rare. Such a tumour is called a cavernous angioma, or, according to Virchow,[1] 'Myoma Telangiectodes.' In these there is an abnormal development of blood-vessels, and the tumour is a spongy mass containing cavities full of blood.

Virchow states that such cavities are lined with endothelium: they are said during life to furnish a loud bruit on auscultation.

Some of these cases appear to be fibro-myomatous, some even sarcomatous[2] from the first, and not fibroids at all. The only case the author has investigated was a degenerating fibroid with hæmorrhage among the fibrous trabeculæ. Sudden hæmorrhage into a so-called cystic fibroid may lead to a rapid increase in its size. Large cystic or degenerating fibroids are not influenced by oöphorectomy: they are generally single and pedunculated. Sometimes a fibroid, which is either intramural or sub-serous, may undergo hæmorrhagic changes, and if in connection with the body of the uterus may give rise to one form of hæmatometra, as in a case of Meredith's[3] (fig. 15). Pyometra also occurs.[4]

Inflammation, Sloughing, and Gangrene.—These changes result from septic infection; sometimes they follow injury or attempts at removal, or supervene on labour or abortion.

Such an infected myoma is œdematous and greyish. It may exhale a sickly odour, or be excessively fœtid or gangrenous. Such tumours are very dangerous, and give rise to severe constitutional symptoms. Sections of an inflamed fibroid resemble sarcomatous tissue very closely, and Bland-Sutton and

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[1] Virchow, *Geschwulstlehre*, vol. iii. p. 195.

[2] Klob, *Wiener Med. Wochenschrift*, 1863, quoted by Haultain, Allbutt and Playfair, *op. cit.* p. 592.

[3] Meredith, 'Hæmatometra associated with a degenerating Fibro-myoma, *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 422; specimen, Museum Royal Coll Surg., Path. Ser. 4604 A (Hæmatometra).

[4] St. Bartholomew's Hospital Museum, No. 2995 Pyometra).

Giles[1] say that there is little doubt that many specimens described as sarcomatous or malignant degeneration of fibroids are of this nature.

Sometimes a sub-mucous fibroid disintegrates when its capsule is injured; septic infection takes place, and the dead fibroid may be present in the vagina as a horribly putrid mass (fig. 66) from which gangrenous shreds of fibrous tissue come away. Sometimes a patient may be cured in this way, at other times fatal infection of the peritoneum takes place through the Fallopian tubes[2] (fig. 67). Herman says such a disintegrated mass may be taken for a placenta.

Sometimes a sub-mucous fibroid which is inflamed and dead may be extruded whole from the uterus into the vagina. A dead sub-peritoneal fibroid may get cast off into the peritoneal cavity and be shut off by adhesions, or local suppuration occurs,[3] and the tumour may eventually be expelled through the abdominal wall by ulceration and suppuration.

Calcification Of Fibroids.[4] (Figs. 68, 69, 70.)—This is a degeneration, and is due to the deposit of lime salts (carbonate and phosphate) in the fibrous tissue. It occurs generally in old fibroids, or in fibroids with a long pedicle and with poor nutrition. Two varieties are found:

(*a*) The **peripheral**, where the deposit is 'en coque,' like an egg-shell, on the surface only.

(*b*) **Interstitial**, where the whole tumour is infiltrated with lime salts. If such a tumour be macerated the calcified mass still exactly resembles the original fibroid, everywhere traversed by trabeculæ (fig. 69).

These calcified tumours,[5] or 'womb-stones,' have been known since the time of Hippocrates. Their cut surfaces can be polished. When found in coffins in old burying-grounds they were imagined to be very large vesical calculi.[6]

In the abdominal cavity they sometimes have very long stalks and may get completely separated[7] from the uterus and may get attached by adhesions to other viscera (fig. 70)

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[1] Bland-Sutton and Giles, *Diseases of Women*, 1st ed., p. 188.

[2] Hurry, *St. Bartholomew's Hospital Reports*, vol. xx.

[3] Herman, *Lancet*, vol. ii. 1894, p. 1346.

[4] Specimen 2996 A, St. Bartholomew's Hospital Museum.

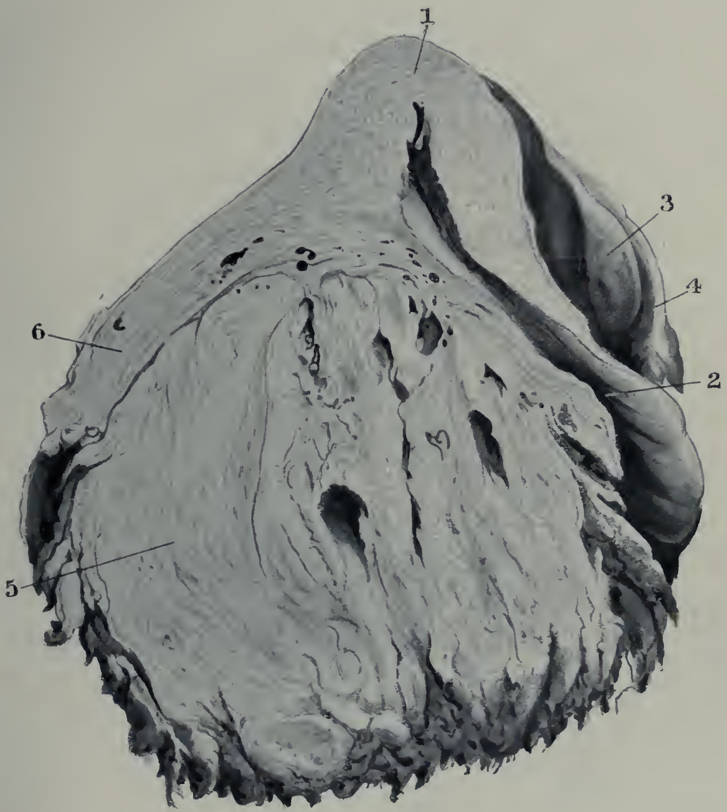
[5] Haultain quotes case wt. = 2 lbs. 5¾ oz., Allbutt and Playfair, p. 585.

[6] Bland-Sutton and Giles, *Diseases of Women*, 1st ed., p. 187.

[7] Specimen 3000, St. Bartholomew's Hospital Museum.

FIG. 66.

SLOUGHING INTRA-CERVICAL FIBROID.

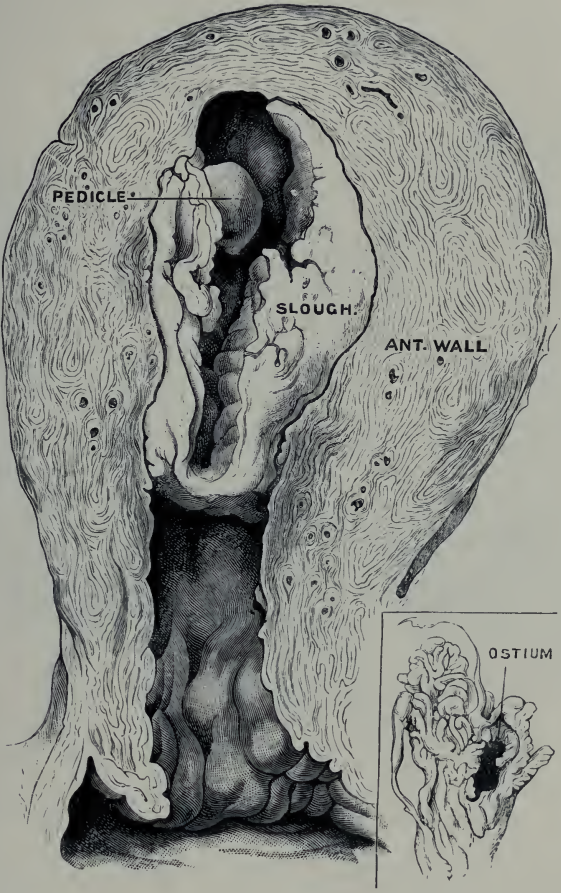


1. Fundus uteri.
2. Canal of cervix displaced by the tumour.
3. Ovary.
4. Fallopian tube.
5. The main mass of the tumour which really occupies the expanded cervix, portions of which still remain above, forming its capsule. The tumour is sloughing below, and large necrotic spaces occupy its central and upper portions.
6. Portion of expanded cervix forming the capsule.

(Specimen 2992 B. Museum, St. Bartholomew's Hospital.)

FIG. 67.

A UTERUS SHOWING THE EFFECTS OF A GANGRENOUS FIBROID.

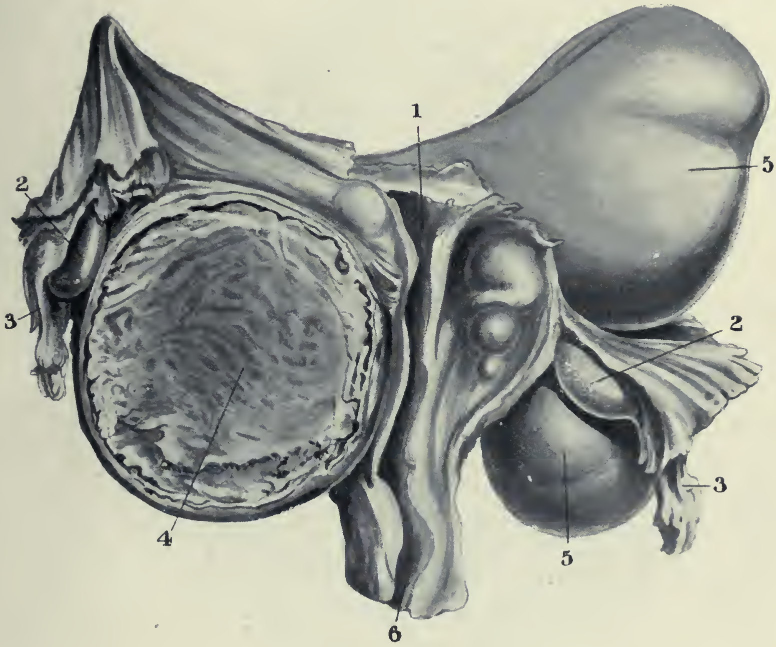


Section of a uterus from which a gangrenous fibroid had been removed by the écraseur. The pedicle is healthy, but the uterine mucosa is in a sloughing condition, which also involved the Fallopian tubes and caused fatal peritonitis. The small figure in the corner represents the open abdominal ostium of the tube.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxii. 1890, p. 172.)

Fig. 68.

CALCIFIED UTERINE FIBROIDS.



1. Fundus of uterus, the cavity of which is very much elongated. In the walls of the uterus are several small fibroids.

2, 2. Ovaries.

3, 3. Fallopian tubes.

4. Large calcified fibroid, partly occupying the right broad ligament.

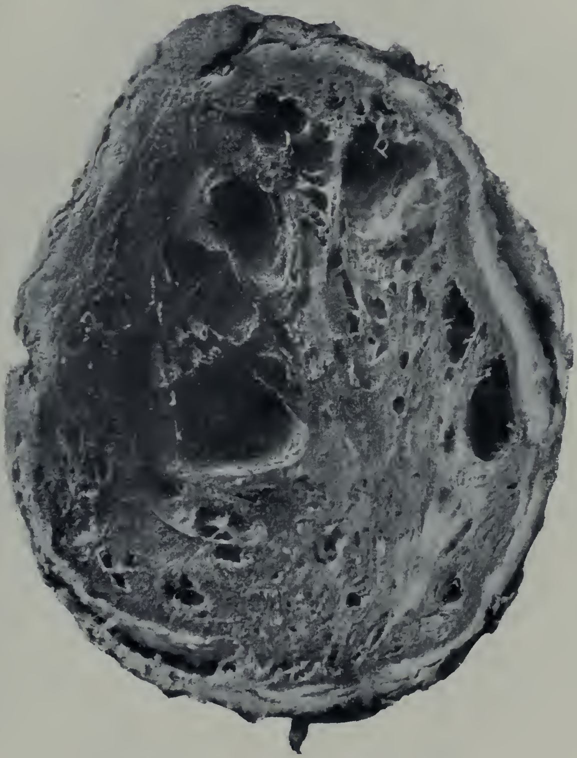
5, 5. Other fibroids, also calcified.

6. Cervix uteri.

(Specimen 2995. Museum, St. Bartholomew's Hospital.)

Fig. 69.

CALCIFIED UTERINE FIBROID.

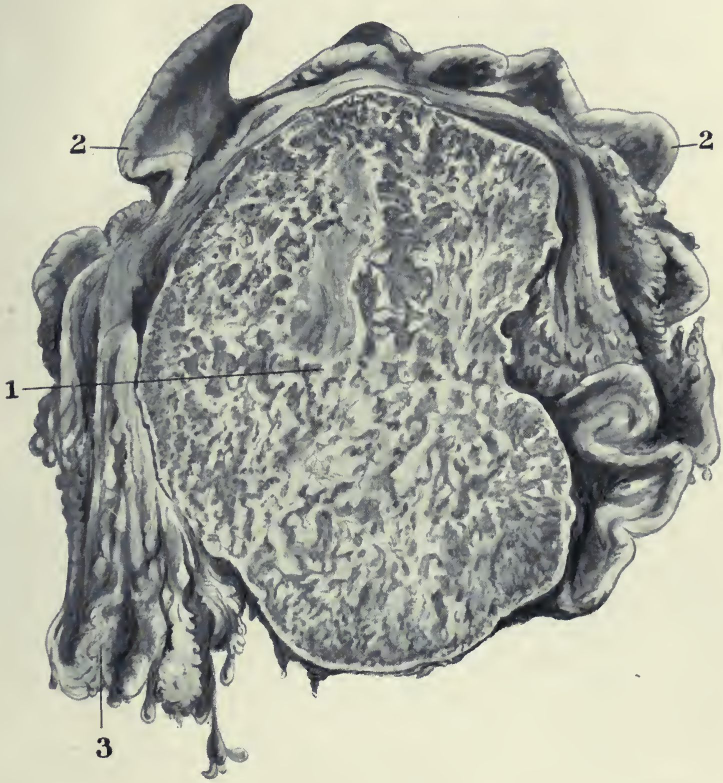


A uterine fibroid which has undergone calcification, chiefly on its outer surface. It has been macerated to show the method of deposition of the lime salts. The tumour is of stony hardness.

(Specimen 2996 A. Museum, St. Bartholomew's Hospital.)

FIG. 70.

A CALCIFIED FIBROID (FOUND LOOSE IN THE ABDOMINAL CAVITY OF AN OLD WOMAN).



Section of a large tumour (7 inches by $3\frac{1}{2}$ inches) taken from the abdominal cavity. It had originally developed in or upon the walls of the uterus, becoming gradually detached; it is everywhere calcified, and all the adjacent viscera are adherent to its surface. Connected to the uterus was a smaller fibroid with a long pedicle which caused death by intestinal strangulation.

1. The main tumour (calcified fibroid), which has no connection with the uterus. The tumour had to be sawn through to expose its interior.	2, 2. Adherent intestine. 3. Omentum.

(Specimen 4642. Museum, Royal College of Surgeons.)

in curious situations, or fall into Douglas's pouch, or get into a hernial sac, or perforate a hollow viscus, or be discharged through the abdominal wall. Some cases of supposed calcified fibroids are extra-uterine (mummified lithopædion) fœtuses: they have been retained in the abdomen as long as fifty years, but the skeleton can always be made out.[1]

Fatty degeneration of a fibroid is exhibited [2] by one specimen in St. Bartholomew's Hospital Museum: it was described by Sir James Paget.

Malignant Degeneration of Fibroids.—This is a subject which, as Bland-Sutton[3] says, is surrounded by clouds of uncertainity. The question is certainly obscure and still needs the most careful observation.

Two distinct classes of cases seem to exist:

(*a*) Those in which a fibroid occurs in a uterus which becomes the seat of cancer,[4] the fibroid being merely involved secondarily. Such cases are quite clear and not altogether uncommon.

Cullen [5] gives some very instructive cases and drawings of uterine fibroids associated with cancer. 'In some the fibroids are small and play a minor rôle; in others the fibroids are very large, and give rise to clinical symptoms overshadowing those due to the carcinoma.' And it appears not to be a very uncommon occurrence, the carcinoma of the body being unsuspected till the uterus is opened after the removal of the fibroid. Sometimes the reverse occurs and the fibroid is not suspected till it causes difficulty in getting the uterus down in vaginal hysterectomy. Recurrences and secondary growths in such cases of course must be carcinomatous, but this is no argument for the malignant degeneration of uterine fibroids. The disease is one of the endometrium, not of the fibroid itself. Cases of cancer of the endometrium in fibroids may give rise to hæmorrhage and foul discharge, rendering the diagnosis between sloughing fibroid and malignant disease difficult till such a uterus is explored.

(*b*) The second class of case is that in which a 'fibroid,' known to have existed for a long time in the uterus, more or less suddenly takes on rapid growth with secondary deposits in various viscera,[1] and in which after death these deposits and the original growth are found to be identical.

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[1] Taylor, *Extra-uterine Pregnancy*, p. 176; Leopold, *Arch. f. Gyn.* vol. xix. p. 210.

[2] No. 3001.

[3] Bland-Sutton and Giles, *Diseases of Women*, 1st ed., p. 188.

[4] Sir J. Williams, *Cancer of the Uterus*, 1888, p. 93.

[5] Cullen *Cancer of the Uterus*, 1900, p. 439. See *ibid* figs 231, 236, 237 244

Great care must be taken in the examination of such cases. It is difficult to follow the transformation of an innocent into a malignant tumour; the microscopical appearances of the original tumour require the closest investigation, and the points of differentiation of a fibro-myoma from a sarcoma are not always easy, and, again, the history of the length of time that the supposed fibroid has existed is open to doubt. Some sarcomata grow slowly, and the degenerative changes in sarcoma, such as sloughing and cystic degeneration, exactly imitate similar changes in fibroids.[2]

The author thinks that it is more probable that such growths are sarcomatous from the first. Probably in microscopic examinations the embryonic nature of the cells and vessels and the absence of a well-marked stroma are points in favour of sarcoma as opposed to fibro-myoma. Champneys[3] says he has never seen a case in which a well-marked fibroid has taken on a malignant growth.

Haultain,[4] speaking of angiomatous changes in fibroids, says such angiomatous changes give rise to fibro-cystic myomata, associated in some instances with an apparently sarcomatous infiltration of the growth proper, an example of which is described by Aslanian, but no reference is given.

Herman[5] says there is some reason to think that fibroids may become the seat of sarcomatous growth, and says he has seen a case in which a bleeding fibroid had dwindled and ceased to bleed for years after 'oöphorectomy,' and then began to grow rapidly; but no mention is made of any microscopical or post-mortem examination.

In two cases operated on by the writer[6] in which the tumour was supposed to be a fibroid, repeated hæmorrhage and the discharge of gelatinous œdematous masses or coagula led to the suspicion of sarcoma. Examination of the curettings proved

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[1] Finlay's case, quoted by Doran, *Trans. Path. Soc.* vol. xli 1890, p. 211.

[2] Routh, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 370, plate v.; also Griffith, *ibid.* p. 232, and plate vi. p. 371.

[3] *Lancet*, vol. i. 1900, p. 150.

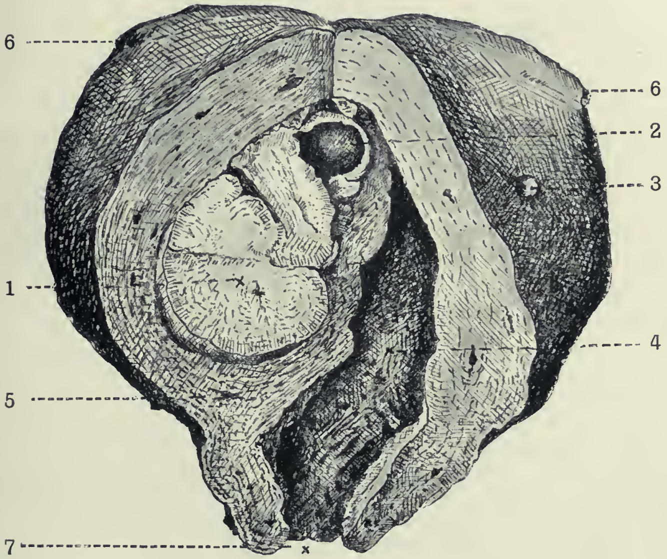
[4] Allbutt and Playfair, *op. cit.* p. 592.

[5] *Diseases of Women*, p. 828.

[6] *Op. cit.*, A. Routh, Griffith. See also article on 'Sarcoma of the Uterus' in this book.

Fig. 71.

SPECIMEN OF SUPPOSED MYXO-SARCOMA OF THE UTERUS.



The specimen was supposed to illustrate malignant degeneration of a uterine fibroid, but there are no microscopic appearances of sarcoma. The specimen is a fibro-myoma undergoing extensive inflammatory and mucoid changes.

1. Tumour, the lower part of which is firm and stands up out of the surrounding tissue. At the upper part it is degenerating and sloughing, and at 2 there is a distinct cyst in the delicate walls. The cyst was filled with mucoid material.
3. Small fibroid, subperitoneal.
4. Enlarged uterine cavity into which the tumour (1) projected.
5. Uterine tissue forming the capsule of the tumour.
6, 6. Fallopian tubes.
7. Cervix uteri.

(A. Routh, 'Trans. Obstet. Soc. Lond.' vol. xli. 1899, p. 367, plate v., drawn by the author.)

one a sarcoma: the uterus was removed, and the patient has since died of sarcomatous deposits in the lungs and viscera. In the other case, after eight curettings between May 1898 and January 1899, the uterus was removed, but the tumour found to be a degenerating cystic fibroid[1] (fig. 71).

Other cases of supposed sarcomatous degenerations of fibroids are reported by Simpson,[2] Ballantyne,[3] Müller,[4] and Whitridge Williams.[5]

The whole question is yet unsettled.

Carcinoma does not occur in a fibroid except by accidental invasion.

A curious tumour is reported by H. Kelly[6] in which a fibroid occupied the anterior wall of the uterus, and was permeated everywhere by structures resembling uterine glands, which appeared to grow in from the mucosa. There was no capsule to the tumour, which gradually blended in the normal uterine muscle. He calls the tumour a benign adeno-myoma. Similar growths also occur in the cervix.[7]

Pathological Results of Uterine Fibroids.—The harm that uterine fibroids do, or may do, is probably overrated. Nothing is commoner than to discover a fibroid in the abdomen causing no symptoms, and exposing the patient to no risk; and as already quoted the risks of fibroids to life, apart from operation, are very small indeed.

As regards pressure and weight, any tumour may cause pressure. A fibroid tumour in the pelvis is very liable to do so, and may cause painful, frequent, or difficult micturition, and such pressure symptoms may lead to retention, cystitis, hydro-nephrosis, and pyelitis, and in rare cases cause death. Pressure symptoms become more marked just before the monthly period. The ureters are sometimes pressed upon by fibroids, but they generally get pushed aside,[8] not involved, in the growth as in cancer. The bladder is sometimes much displaced by fibroids. The bowel is rarely interfered with; there may be painful or difficult defæcation, but rarely obstruction. It is more likely that in fibroids the bowel is weakened from interference with its circulation, innervation, and nutrition rather than from direct pressure (Champneys).

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[1] A. Routh, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 367.

[2] A. R. Simpson, *Contributions to Obstet. and Gyn.* p. 240. Edin. 1880.

[3] Ballantyne, *Edin. Med. Jour n.* 1884.

[4] Müller, *Arch. f. Gyn.* vol. xxxix. 1891.

[5] Whitridge Williams, *Amer. Journ. Obstet.* vol. xxix. 1894, p. 721.

[6] H. Kelly, *Operative Gynæcology*, vol. ii. p. 387.

[7] Herman, 'Follicular Hypertrophy of the Cervix,' *Trans. Obstet. Soc. Lond.* vol. xxii. 1880, p. 270; Blacker, *ibid.* vol. xli. 1899, p. 374. See chapter on 'Cancer of the Uterus' in this book.

[8] Champneys, *Lancet*, vol. i. 1900, p. 148.

Varicose veins of the legs or abdomen are rare; they are much commoner with cancer. Interference with pelvic veins by fibroids is not common, but piles do occur, while at times the lividity of the vulva and vagina resembles that of pregnancy.

Fibroids sometimes cause neuralgia by pressure on nerves of pelvis. Much of the pain with fibroids is due to painful uterine contractions intensified at the monthy period. This is the so-called fibroid dysmenorrhœa. Such pain occurs with fibroid uteri which are only very slightly enlarged, and is not due to pressure, although in large fibroids, as before mentioned, pressure on pelvic nerves may occur. An inflamed fibroid with surrounding peritonitis may also be painful. Pain is urged by some as a correct indication for removal if the objective signs of suffering are great, but the personal equation [1] of pain is different in all. The amount of pain a patient says she suffers is one thing, the results of pain observed is another.

Actual sloughing and gangrene [2] of the pelvic soft parts may occur from the pressure of incarcerated tumours.

Owing to increased intra-abdominal pressure and the increased blood supply necessary for a large tumour the heart [3] may be hypertrophied as in pregnancy.[4]

Fibroids and Hydro-peritoneum.—Free fluid with fibroids is excessively rare. This fact is somewhat astonishing, as it would appear that some tumours would cause irritation; still it is very unfrequent. Cases have been observed which were due to venous pressure, morbus cordis, or nephritis. In others the hydro-peritoneum appears to be due to some chronic peritonitis by which the tumour is affected, owing to its local degeneration. In other cases no cause can be discovered.

Fibroids and Pregnancy.[5] (Fig. 72.)—Fibroids are said to

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[1] Champneys, *Painful Menstruation*, p. 18.

[2] Haultain, Allbutt and Playfair, p. 578.

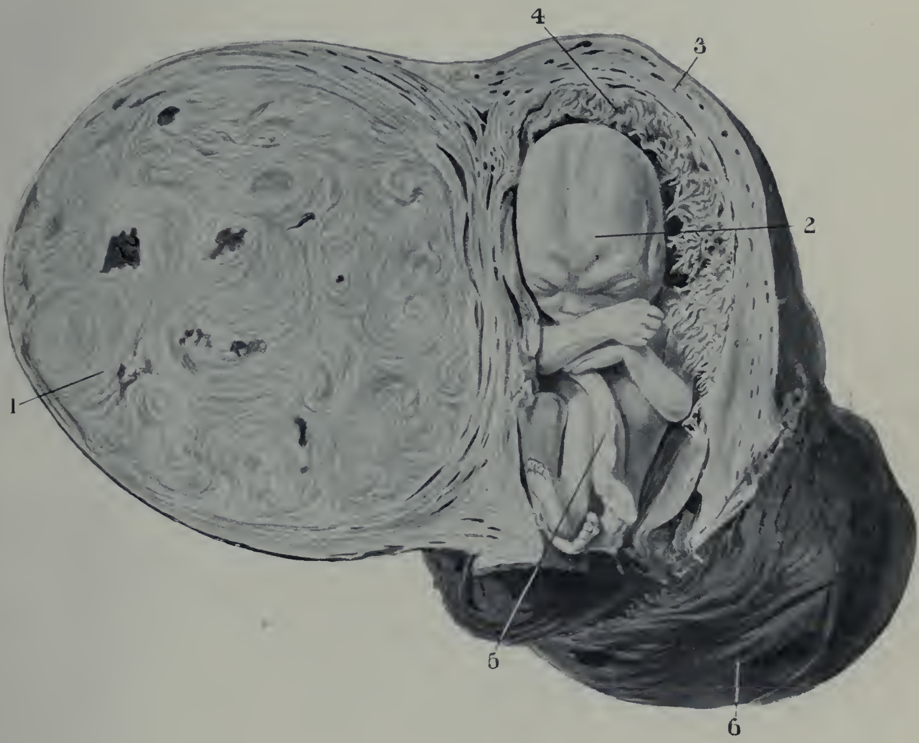
[3] *Ibid.* p. 578.

[4] See also Thomas Wilson, 'Relation of Organic Affections of the Heart to Fibro-myoma of the Uterus,' *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 176.

[5] Cripps, *Ovariotomy and Abd. Surg.* p. 501, plate xviii; specimen 3200 A, St. Bartholomew's Hospital Museum; Doran, 'A Case of Hysterectomy for Uterine Fibroid in the fifth month of Pregnancy; Recovery, with references to recent cases,' *Lancet*, vol. i. 1901, p. 621; *id.* 'On Myoma and Fibro-myoma of the

FIG. 72.

UTERINE FIBROIDS AND PREGNANCY.



Section of a uterus and fibroid tumour. The uterus contains a fœtus at about four and a half months.

1. Large fibroid growing from the side of the uterus.
2. The fœtus at about fourth and a half months.
3. Wall of uterus, showing large blood-sinuses.
4. Placenta.
5. Umbilical cord.
6. Another mass of fibroids which completely blocked the pelvis, obstructing delivery.

(Specimen 3090 A. Museum, St. Bartholomew's Hospital. Cripps, 'Ovariotomy and Abdominal Surgery,' p. 501.)

cause sterility, but they do not always do so; possibly the question is rather that when a fibroid exists that can be diagnosed the patient has already reached the period of relative sterility. Great bleeding tends, of course, to prevent conception, and the thickened endometrium[1] of fibroids is also adverse to it. Yet women with fibroids do conceive, and go to term, and are frequently delivered naturally. Sometimes abortion occurs, with risk of septic infection of the tumour, and death,[2] or incarceration of the uterus below the brim as it enlarges. At full time hopeless obstruction to labour may occur, but it is generally the pelvic, cervical, or broad ligament fibroids that are so dangerous,[3] not the large tumours above the brim. Still, the dangers of fibroids complicating pregnancy, labour, and the puerperium may be of the most grievous nature.[4] A fibroid uterus is liable to inertia and the risks of post-partum bleeding. During pregnancy the fibroids enormously increase; the muscle cells hypertrophy, and the tumour becomes soft, while after delivery involution of the uterus and fibroid may occur, but it is not always the case. Infection after parturition in such cases may lead to sloughing of the fibroid, septic endometritis, and, not infrequently, death. Sometimes a sub-mucous fibroid may be present in front of the fœtus during labour, or may be enucleated[5] and expelled at the time. Fibroids of the posterior wall of the uterus are more liable to cause obstruction to labour than those situated anteriorly (Doran, Phillips).

If the placenta be situated over the capsule of a large sub-mucous fibroid, very dangerous, or even fatal, hæmorrhage may occur if a large sinus be opened.

Uterus,' *Trans. Obstet. Soc. Lond.* vol. xxx. 1888, p. 410, in which Doran demonstrates the marked hyperplasia occurring in the muscle cells of a myoma associated with pregnancy; John Phillips, 'Treatment of Pregnancy and Labour complicated by Fibro-myomata,' *King's College Hospital Reports*, vol. vi., 1900, p. 27; Armand Routh, *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 244.

[1] Herman, *Diseases of Women*, p. 639.

[2] Haultain, Allbutt and Playfair, *op. cit.* p. 593.

[3] See also Griffith, 'Labour obstructed by a large Fibroma of the Ovary,' *Trans. Obstet. Soc. Lond.* vol. xxxiii. 1891, p. 140, and plate, p. 144; and Bland-Sutton, 'Surgery of Pregnancy and Labour complicated with Tumours,' *Lancet*, vol. i. pp. 16, 23, 1901. See also chapter in this book on 'Diseases of the Ovaries.'

[4] Bland-Sutton, 'The Surgery of Pregnancy and Labour complicated by Tumours,' *Lancet*, vol. i. 1901, p. 452; also Champneys, *St. Bartholomew's Hospital Reports*, 1877.

[5] Dakin, 'Fibro-myoma spontaneously enucleated during Labour,' *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 105.

Fibroid of the uterus may be complicated by extra-uterine pregnancy[1] (figs. 35, 36, 37).

Fibroids have sometimes caused inversion at the time of delivery. Fibroid polypi are sometimes associated with chronic inversion.[2]

Fibroids of the Broad Ligament.[3] (Fig. 73.)—These tumours have till lately received little notice, and possibly represent a class of fibroids distinct from those of the uterus, and, at all events, not primarily of uterine origin.

It is of course well known that many fibroids that arise in the uterus, either in the body or the cervix, may grow out and burrow between the layers of the broad ligament, producing great distortion and displacement of the pelvic contents, often with serious pressure symptoms.

Tumours of this sort are of course uterine in origin, but true fibroids of the broad ligament probably arise from the muscular fibres which occur in the broad ligament immediately beneath the peritoneum, and which, as previously mentioned, are sometimes well marked (the so-called platysma): this is directly continuous with the musculature of the uterus. In the same category come the round ligament and ligament of the ovary, which may themselves give rise to fibroid tumours.

Arising as such, fibroids of the broad ligament are essentially sub-serous, and have primarily no connection with the uterus: they burrow deeply amid the layers of the broad ligament, and may even strip up the peritoneum of the anterior abdominal wall, or that covering the sacrum and rectum. They have little or no connection with the uterus except by

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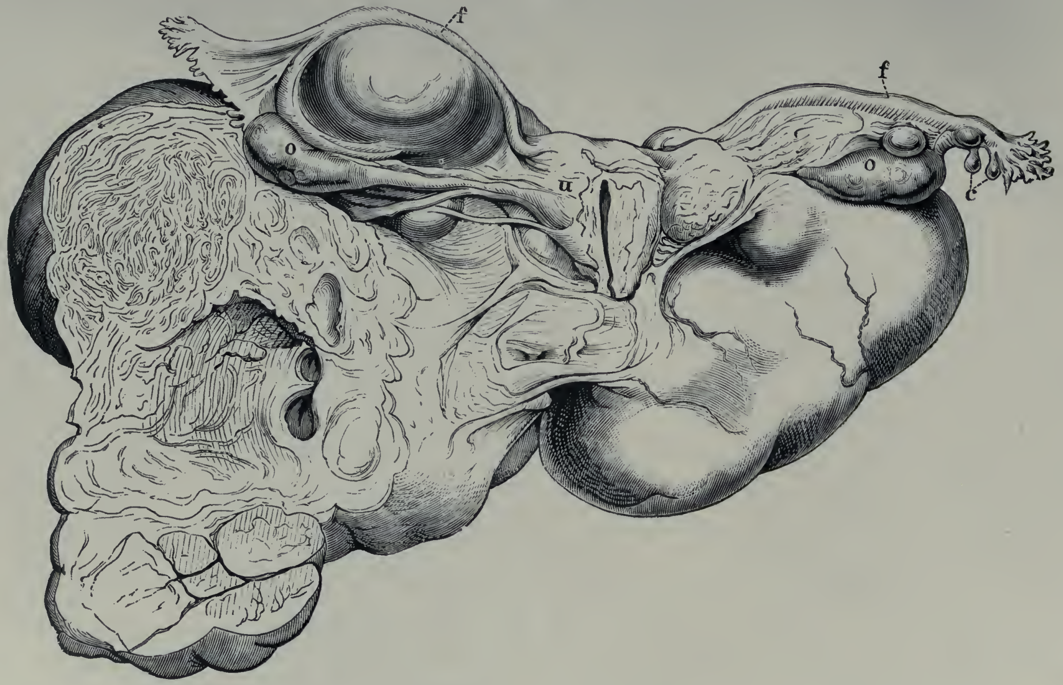
[1] Cullingworth, 'Ectopic Gestation complicated by Fibro-myomata,' *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 285.

[2] Specimen 2970, St. Bartholomew's Hospital Museum.

[3] Doran, 'Fibroid of the Broad Ligament weighing 44½ lbs. removed by Enucleation; Recovery, with table and Analysis of 39 cases,' *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 173; Roberts, 'Large Retro-peritoneal Fibroid undergoing Suppuration,' *ibid.* p. 213; Meredith. *ibid.* vol. xxix. 1887, p. 249; Cullingworth, *ibid.* vol. xxxvii. 1895, p. 222; Sänger, *Arch. f. Gyn. u. Geb.* vol. xvi. p. 258; Bayard Holmes, *Journ. Amer. Med. Assoc.* vol. x. 1881, p. 191; Lang, 'Des Tumeurs solides primitives des ligaments larges,' Thèse de Nancy, 1892, p. 86; R. B. Hill, *Amer. Journ. Obstet.* Nov. 1899; Bellfinger, *Inaug. Dissert.* Würzburg, 1887; Bantock, *Brit. Gyn. Journ.* vol. iii. 1887, p. 493; Cripps, *Ovariotomy and Abdominal Surgery*, p. 491; Gross, *Congrès Français de Chir.*, 6^e Sess. 1892, p. 249; Bland-Sutton, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 298, and *Surgical Diseases of the Ovaries and Fallopian Tubes*, 2nd ed., p. 348.

Fig. 73.

MYOMA OF THE MESOMETRIUM.



f f. Fallopian tubes. o. Ovaries. u. Uterus. c. Cysts of the tube.

Broad-ligament fibroids may grow very rapidly and reach a great size. They cause the most remarkable displacement of the uterus, appendages, and peritoneum, especially involving the anterior layer of the mesometrium.

(Bland-Sutton, 'Diseases of the Fallopian Tubes,' new edition, p. 348.)

apposition or slight adhesion, and the uterus itself is generally not enlarged or altered in any way, but is merely displaced forwards and upwards or to one side by the growth. Such growths often attain very large dimensions[1] and for this reason cause ill-health. They are generally sessile and fixed. They are composed of dense fibrous tissue like uterine fibroids, and are nourished by a false capsule composed of layers of the broad ligament. Their nutrition is poor, and they are liable to degeneration and suppuration.

In the case reported by the writer the tumour was practically converted into a thick-walled abscess cavity[2] containing over thirteen pints of pus. Large fibroids of the broad ligament have been removed from women under thirty;[3] they are apt to grow rapidly, and if large cause great displacement of the peritoneum, pain, discomfort, and pressure symptoms. Flooding according to Doran does not occur, but patients are liable to be often sickly and anæmic. They are very difficult to remove, and are supplied by numerous vessels from the capsule, which give great trouble at the time of operation. Retroperitoneal hysterectomy is the best procedure (Doran). Sometimes it may be possible to enucleate a tumour of this sort from the broad ligament without sacrificing the uterus.

Bland-Sutton[4] thinks that some tumours of the mesometrium may arise in the cervix and spread thence into the broad ligament; others are ordinary fibroids of the fundus which push their way between the layers of the broad ligament in the same way; while quite distinct from these is the variety described above as fibroids arising in the broad ligament. Bland-Sutton's case weighed twenty-two pounds, and on section resembled rather a spindle-celled sarcoma than a fibromyoma.

Tumours of this sort may occur in other situations than the broad ligament, and according to Bland-Sutton[5] generally take on a shape resembling a football: they may arise in the retro-peritoneal tissues beneath the liver or in the lesser bag of the peritoneum. Such tumours can be easily shelled out of their capsule: they are invariably globular, and may attain a great size, and conform in type to very slow-growing spindle-celled sarcomata (Bland-Sutton); but, as before pointed out, the greatest difficulty arises in distinguishing under the microscope sections of a fibroma from spindle-celled sarcoma, and, clinically at all events, some of these retroperitoneal growths appear innocent.

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[1] Doran, 44½ lbs., *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 295, vol. xli. 1899, p. 173; Roberts, 38 lbs., *ibid.* vol. xli. 1899, p. 215. [2] Roberts, *op. cit.*, p. 213.

[3] In Doran's thirty-four cases where age was given, 17·6 per cent. occurred in women under thirty. Table, *loc. cit.* p. 188.

[4] 'Tumour of the Mesometrium weighing 22 lbs.,' *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 298.

[5] Bland-Sutton, *op. cit.*, p. 300. Marmaduke Shield, 'Large, Solid Tumour removed with success from the Retro-peritoneal Space,' *Med. Chir. Trans.* vol. lxxx. 1897, p. 205.

Fibroids of the Round Ligament.—These are very rare, and may occur either in the inguinal canal, outside the abdomen, or within the abdominal cavity.[1] They are generally solid, firm tumours giving rise to little pain or trouble of any sort. They are said to increase in size before menstruation. Their growth is slow and they are rarely diagnosed before operation.

Within the abdomen similar fibroid masses may occur, growing from the round ligament; but the diagnosis cannot be made before operation. The only interest of such tumours is their seat of origin.

Fibroids of the Anterior Abdominal Wall are of interest in that they are sometimes taken to be intra-peritoneal tumours, but they are in the wall and are fixed by muscular contraction. They are rare, and appear to grow from retro-peritoneal tissues or the transversalis fascia, and are closely connected with the peritoneum. They are firm, smooth tumours, generally single, and are never in the middle line:[2] they are composed as a rule of white fibrous tissue, though some are sarcomatous, and are subject to the same degeneration as fibroids of the uterus. These tumours are, for some unknown reason, far more frequent in women than in men. In Grätzer's series of thirty-eight cases only five occurred in males.[3] They have been observed to increase during pregnancy.[4]

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[1] Herman, *Diseases of Women*, p. 867, who also quotes Sänger, *Arch. f. Gyn. u. Geburtsh.* vol. xxi. p. 279.

[2] Herman, *op. cit.* p. 833.

[3] Doran, 'Fibro-sarcoma or "Dermoid Growth,' of the Abdominal Wall,' *Trans. Med. Soc.* vol. xiv. 1891, p. 337, with literature.

[4] *Id.* 'Fibroma of the Abdominal Wall; Increase during Pregnancy,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 42.

CHAPTER XIII

ON UTERINE POLYPI

Uterine Polypi are generally of the following varieties:

1. Fibrous Polypi.
2. Mucous Polypi.
3. Placental or Fibrinous Polypi.
4. Malignant Polypi.

Fibrous Polypi.—These are most probably extruded hard fibroids, and are the same as those which are first met with as intramural fibroids of the body of the uterus. They grow inwards, pushing the endometrium before them. They may possibly arise from the unstriped muscle of the deeper layers of the endometrium itself.[1]

The tumour, which at first becomes sub-mucous, is, it is said, gradually pushed forwards into the cavity of the uterus, and becomes more or less stalked (fig. 74): it reaches the cervix, which after a time dilates and allows the polypus 'to be born,'[2] so to speak, into the vagina; eventually it may even protrude at the vulval orifice.

The above process is that which is always described, but it is very doubtful if all the real stages have been accurately traced from the embedded tumour which becomes a sessile tumour and then a polypus.[3] Matthews Duncan says he doubts the truthfulness of the description.[4] Some polypi of this nature when they have reached the cervix, but have not yet protruded through it, can only be felt during menstruation; after this is over they retreat and the cervix closes: hence the name *intermittent polypus.*[1]

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[1] See paragraph on 'The Origin of Uterine Fibroids' in this book.

[2] Matthews Duncan, *Clin. Lectures*, 4th ed. p. 318.

[3] Haultain, article 'Benign Growths of the Uterus' Allbutt and Playfair, *op. cit.* p. 567, makes out two varieties:

(*a*) The 'encapsuled,' which arises from middle layers of the muscle.

(*b*) The 'free' variety, developed from internal layers of uterine muscle closely connected with the superimposed mucosa, which forms the false capsule, and which nourishes the tumour.

[4] Matthews Duncan, *Clin. Lectures*, p. 208, 4th ed.

So long as the tumour is within the uterus it is covered with the columnar ciliated epithelium of the mucosa and a certain amount of glandular elements, the thickness of the endometrium being variable, but when the tumour comes into the vagina the epithelium is very poorly nourished and œdematous, the surface of the tumour is frequently purple, of very low vitality, and the slightest injury may cause it to slough. This is due to the poor nutrition and feeble blood supply of these tumours, as instanced by the slight hæmorrhage which occurs when they are cut through at operation.[2] Fibroid polypi have practically no capsule, except at their base; in this they appear to differ very much from intramural fibroids, in which all stages of enucleation have been observed, and it seems more probable that the origin of fibroid polypi is not the same, but rather, as before stated, from the deeper muscular layers of the endometrium.

In some fibroid polypi which are in the vagina the surface of the tumour is covered with well-marked squamous epithelium: it is probably due to friction and exposure, much the same as in an everted split cervix, where the same change occurs, the columnar epithelium of the cervical canal being replaced by squamous cells over the exposed areas. Fibroid polypi cause great hæmorrhage and profuse catarrh of the endometrium. As a rule, as long as the polypus is within the uterine cavity the bleeding is menorrhagic in type, but if in the vagina the loss becomes continuous.

These polypi cause painful uterine contractions, especially as the tumour is passing the cervix. They are analogous to expulsive labour pains.

Fibroid polypi vary in size; they are generally elongated in shape and they do not exceed the size of a fœtal head or they could not descend into the pelvis, though œdema may increase their size when they are once in the vagina, and in this situation their pedicle is so stretched, and their nutrition so low that, besides being œdematous, they may soften, disintegrate,

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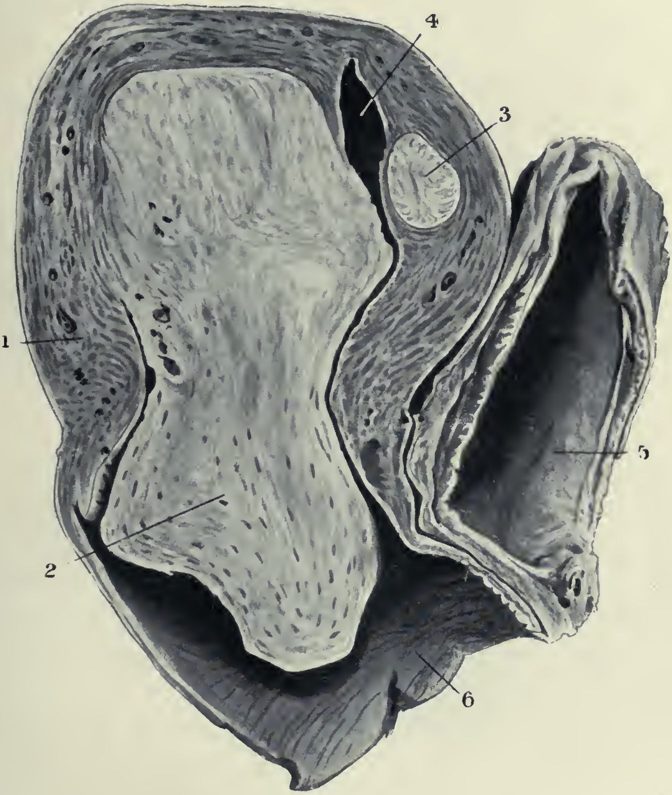
[1] Matthews Duncan, *op. cit.* p. 319.

[2] Matthews Duncan, *op. cit.* p. 211, explains the absence of hæmorrhage in the removal of fibrous polypi as being due to the powerful retraction of the pedicle.

Fig. 74.

FIBROID POLYPUS OF THE UTERUS.



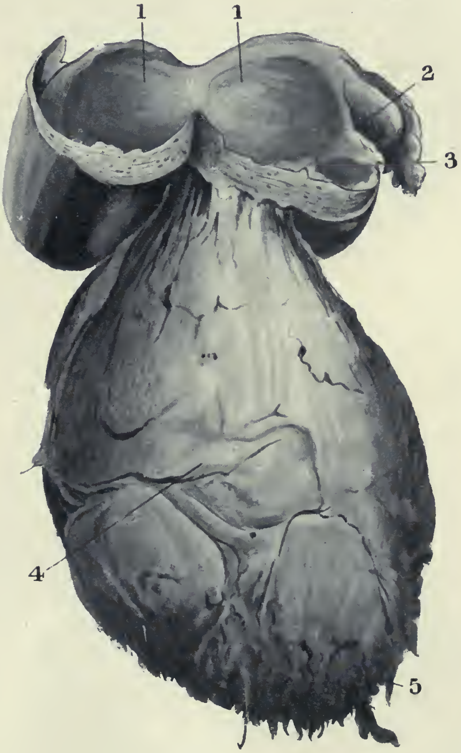
A uterus the cavity of which is filled with a large polypoid tumour. It is attached by a broad base to the fundus and posterior wall; it projects through the cervix into the vagina, and measures 5 inches in length. It had caused metrorrhagia for several years.

1. Uterine wall containing large sinuses.
2. The fibroid polypus.
3. Small fibroid in anterior wall of uterus.
4. Cavity of uterus.
5. Bladder.
6. Vagina.

(Specimen 4615 A. Museum, Royal College of Surgeons. Doran, 'Brit. Med. Journ. vol. i. 1894, p. 1233.)

FIG. 75.

LARGE FIBROID POLYPUS (SLOUGHING).



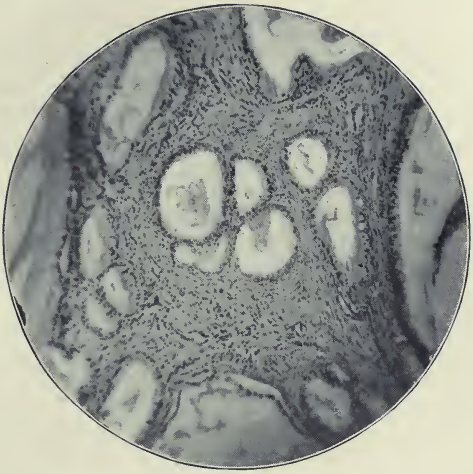
A large intra-uterine polyp with a broad base, growing from the body of the uterus. The uterus has been divided and the margins of the cervix turned upwards. The tumour extended into the vagina, and was undergoing necrotic changes at its lower part.

1, 1. Fundus uteri.
2. Ovary.
3. Fallopian tube.
4. Main mass of the tumour which protruded from the cervix and filled the vagina.
5. Portion of the tumour which is sloughing.

(Specimen 2973. Museum, St. Bartholomew's Hospital.)

FIG. 76.

SIMPLE MUCOUS POLYPUS OF THE CERVIX.

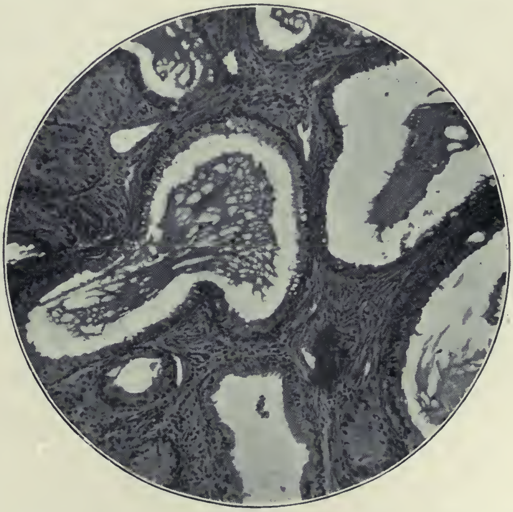


Section of a small mucous polyp which protruded from the cervix uteri. The growth shows spaces lined with a single layer of columnar epithelium, identical with that lining the glands of the cervix. Many of the spaces contain mucin. The stroma in places shows myxomatous changes.

(Microphotograph by C. H. Cosens.)

FIG. 77.

SIMPLE POLYPOID GROWTH OF THE CERVIX.



Section of a polypoid growth which sprang from the cervix. The growth was soft and vascular, and resembled an ordinary mucous polypus, but it was channelled everywhere by cystic spaces containing mucin. It is an ordinary cystic adenoma, the spaces being lined with a regular layer of columnar epithelium resembling that of the glands of the cervix. (High power.)

(Microphotograph by C. H. Cosens.)

and die (fig. 75), and septic organisms reaching the dead tissue gangrene results, with a horribly fœtid discharge, the patient becoming poisoned by septic absorption. They may even slough completely off. Sometimes in large tumours projecting into the vagina pressure may destroy the opposing surfaces or epithelium, and the most curious and puzzling adhesions take place to the vaginal wall.

The traction of a fibroid polypus may produce inversion[1] (such inversion is of course chronic). This is to be thought of in removing such growths, or a hole may be made in the uterine wall. Care must be taken in the diagnosis of a fibroid polypus from an inverted uterus.

Prolapse with fibroid polypus also occurs. Such a case is figured by Herman,[2] but in this case the tumour grew from the cervix, and the traction would not be likely to produce inversion. In these cases the uterus only is dragged on and descends, the bladder and rectum being in their normal situation. They are rare.

Cervical Fibroids[3] are sometimes mistaken for fibroid polypi, but these grow from the cervix and are encapsuled by its tissues. Cervical fibroids have no stalk and the slit-like orifice of the expanded cervix can be felt to one side.

Cysts of Cervix—or cystic adenomata, sometimes appear stalked. Some growths of a polypoid nature from the cervix are sarcomatous: they are commoner in children than in adults.[4]

Mucous Polypi.—These may grow either from the cervix or body of the uterus: they are commoner in the cervix. They are stalked adenomata and resemble the normal mucous membrane, and are made up of uterine glands forming alveoli, which are often more or less irregular, dilated, and tortuous, and lined with a beautiful single layer of columnar epithelium (fig. 76). The stroma consists of loose connective tissue often highly myxomatous, and the glands themselves are often filled with mucin, and definite cystic cavities sometimes exist (fig. 77). Some mucous polypi riddled with these cyst-like cavities are called *channelled polypi* (fig. 78). If the stroma is more or less dense instead of myxomatous, they are termed *fibro-cellular polypi.*

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[1] Matthews Duncan, *op. cit.* pp. 210, 319.

[2] Herman, *Diseases of Women*, p. 115, fig. 25.

[3] For 'Cervical Fibroids' see chapter on 'Uterine Fibroids'; also Bland-Sutton, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 160.

[4] See chapter on 'Cancer of the Uterus.'

Large stalked ovoid growths consisting of glandular structure like cervical glands occur with pregnancy, and are known as *follicular hypertrophy of the cervix.*[1]

The hypertrophic polypus of Barnes[2] is a piece of cervix which has been separated by a laceration on each side, and which then gets enlarged by chronic inflammation. It may even have a distinct pedicle.

The common mucous polypus is generally about the size of a pea, though some are considerably larger. They are elongated in form, reddish in colour, hanging from the cervix by a stalk or just protruding at the external os. They are usually quite soft and often difficult to feel. Mucous polypi are frequently multiple, and are associated with the flat adenomatous condition called erosion. Frequently there is also cervical catarrh and chronic inflammation of the cervix. The stalk of a mucous polypus may be of some length and contain small vessels which may bleed considerably on removal. Mucous polypi also occur in the body of the uterus, and are not at all uncommon with fibroids as previously stated.[3] They are pedunculated adenomata and are practically identical with the mucous polypi of the cervix, only they are often larger and flattened by pressure. Mucous polypi that are long exposed in the vagina get covered with a cap of squamous epithelium, much in the same way as fibroid polypi.

Their relation to cancer is not proved, but some growths taken at first to be mere mucous polypi may be proliferating portions of cancer and should be examined microscopically. Sir John Williams[4] reports a case of cancerous squamous epithelium found on the lower end of a mucous polypus of the cervix, and he says, further, that uterine polypi in advanced age are apt to be malignant, and whenever removed they should be examined as to their true character.

The variety of adenoma in which the fibrous element predo-

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[1] Herman, 'Follicular Hypertrophy of Cervix,' *Trans. Obstet. Soc. Lond.* vol. xxii. 1880, p. 270.

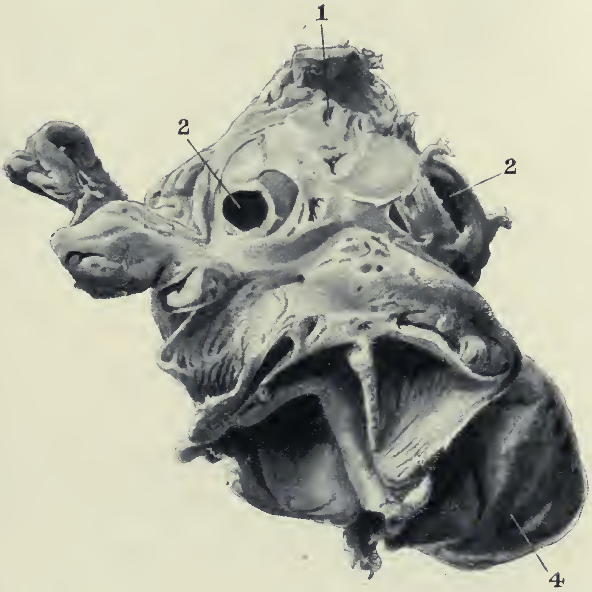
[2] Herman, *Diseases of Women*, pp. 374, 454.

[3] They may also occur with cancer of the cervix (Roberts, *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 267).

[4] Sir J. Williams, *Cancer of the Uterus*, p. 72; Harveian Lectures, 1886.

Fig. 78.

CHANNELLED MUCOUS POLYPUS OF THE CERVIX.



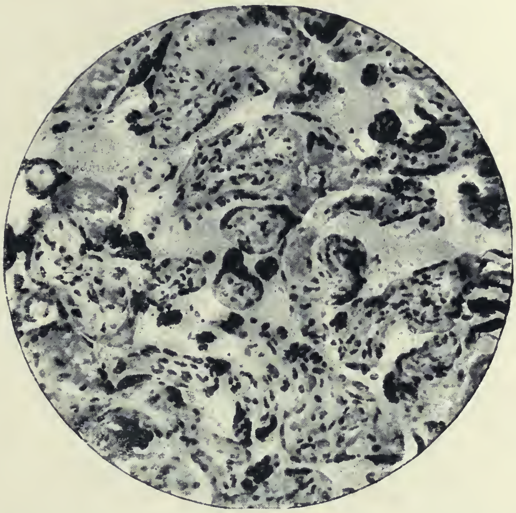
A large soft polypus removed from the cervix uteri. The growth is riddled with cavities of irregular size, which were filled with thick mucus. On section it proved to be a cystic adenoma. It is very vascular.

1. Pedicle.
2, 2. Large cystic spaces with which the whole tumour is channelled.
4. More solid portions of the tumour.

(Specimen 2967 B. Museum, St. Bartholomew's Hospital.)

FIG. 79.

CHORIONIC VILLI.



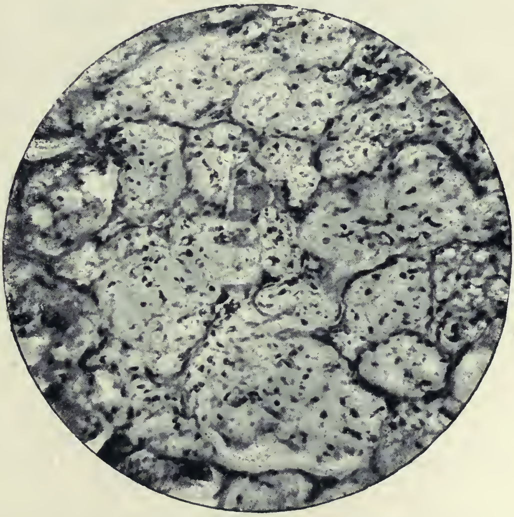
Chorionic villi from an eight months' placenta, retained two to three weeks (× 130).

The section shows a part of the placenta from which the maternal circulation has not been cut off. There is fresh blood in the intervillous spaces, and some of the villi show blood-containing capillaries.

(Eden, 'Trans. Obstet. Soc. Lond.' vol. xxxviii. 1895, p. 366.)

FIG. 80.

CHORIONIC VILLI.



Chorionic villi from an eight months' placenta, retained two to three weeks (× 130).

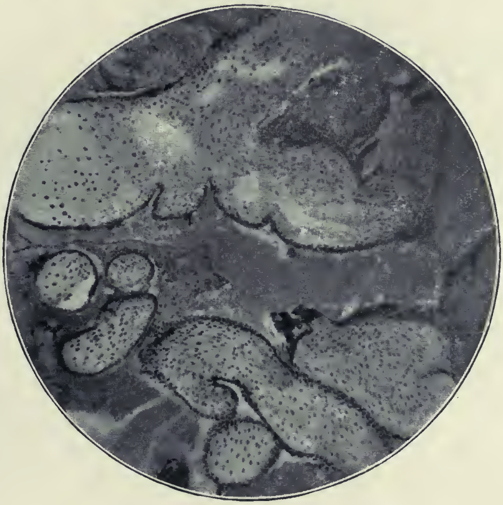
Another portion of the same placenta as in fig. 79.

It shows a part of the placenta from which the maternal circulation has been cut off. The villi are closely packed together, obliterating the intervillous spaces; most of them have lost their epithelial covering, the stroma is degenerating, and the capillaries have entirely disappeared.

(Eden, 'Trans. Obstet. Soc. Lond.' vol. xxxviii. 1895, p. 372.)

Fig. 81.

PLACENTAL POLYPUS.

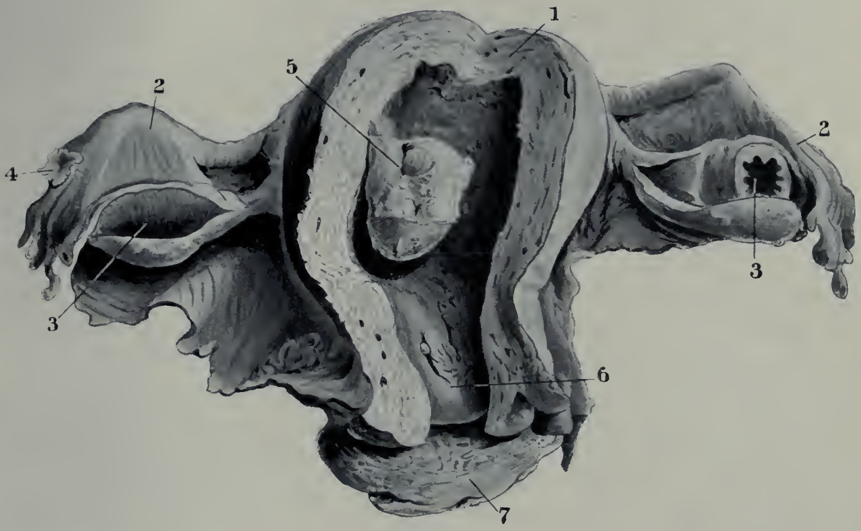


Section of a placental mass which had been retained seven months in a uterus after an abortion. The chorionic villi are well preserved, and still retain their epithelial covering (syncytium) in good preservation; in some places syncytial buds are seen. The stroma of the villi has undergone degenerative changes, and the villi themselves are embedded in old fibrin and altered blood-clot.

(Microphotograph by C. H. Cosens.)

FIG. 82.

PLACENTAL POLYPUS.



A uterus much enlarged and laid open to show a soft, pedunculated, very vascular placental polypus, growing from the inner wall midway between the left Fallopian tube and the os internum. It was composed of a mass of placental tissue.

1. Body of uterus.	5. Placental mass.
2, 2. Fallopian tubes.	
3, 3. Ovaries.	6. Cervix.
4. Accessory ostium near extremity of left tube.	7. Vagina.

(Specimen 4659. Museum, Royal College of Surgeons. Doran, 'Trans. Obstet. Soc. Lond.' 1895, vol. xxxvii. p. 229.)

minates is described by Haultain[1] under the heading of fibro-adenoma, and is primarily a localised hypertrophy of the normal mucous membrane. The fibrous tissue is in excess; the glands are large and form canals which permeate the tumour in all directions: they seem to be identical with the channelled polypus of the cervix. They contain much embryonic and cellular tissue at the periphery immediately beneath the epithelium, but do not seem to be malignant.

Fibrous Papilloma.—This is a rare form of simple polypus growing from the uterine mucosa, and appears as a fibrous tumour of papillary form covered by a single layer of epithelium. There may be offshoots from the original tumour, and the tumour may have a cauliflower or even villous appearance: they appear to be benign, or are allied to epithelioma,[2] though Haultain says microscopic examination and clinical observation at once disprove such an affinity.[3] The differences in structure and varied microscopical appearances exhibited by these growths from the endometrium are most puzzling.[4]

Placental and Fibrinous Polypus.—These are mere names for pieces of placenta, decidua, or chorion (figs. 79, 80, 81) retained within the uterine cavity, which have a tough vascular adhesion to some part of the uterine wall. Blood-clot is deposited upon the mass, for the mass causes bleeding, and the bleeding gives rise to further clotting over the retained mass. This is the so-called placental polypus (fig. 82). The fibrinous polypus is merely blood and fibrin of various ages retained in the uterus and adherent where the chorion was attached. Haultain describes a fibrinous polypus that occurred apart from pregnancy.[5] Whatever be the origin of the retained mass, it keeps up hæmorrhage till it comes away or is removed.

It has yet to be proved that such retained products of conception can grow after the expulsion of the ovum or fœtus, and it is equally doubtful if such (fœtal) structures can give rise to new growth.[6]

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[1] Allbutt and Playfair, *op. cit.* p. 606.

[2] See, however, Routh, 'Malignant Papilloma of Uterus,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 5.

[3] Haultain, 'Benign Growths of the Uterus,' Allbutt and Playfair, *op. cit.* p. 607.

[4] See chapters on 'Cancer of the Body of the Uterus' and 'Endometritis,' where the subject is more fully discussed.

[5] Haultain, Allbutt and Playfair, *op. cit.* p. 609; and *Trans. Obstet. Soc. Edinburgh*, 1893.

[6] See *Deciduoma Malignum*, in chapter on 'Cancer of the Uterus.'

Malignant Polypi.—These are practically pedunculated pieces or portions of either cancer or sarcoma of the cervix or body; as such they should be classed, and not under the heading of polypi at all. It is of course possible that such prolongations may assume a polpyoid form, sarcoma of the cervix certainly may do so. Some so-called mucous polypi are malignant, and carcinomatous or sarcomatous masses growing from the body of the uterus may be felt at the cervix or protrude into the vagina.

Some polypi of the endometrium are certainly malignant, and the greatest difficulty arises in the diagnosis between 'benign' and 'malignant' adenoma; in fact, there is no way[1] of distinguishing such except by microscopical examination and by observing the rapidity of return after removal.[2]

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[1] Herman, *Diseases of Women*, p. 351.

[2] See also chapters on 'Endometritis' and 'Cancer of the Body of the Uterus.'

CHAPTER XIV

ON DISEASES OF THE OVARIES

THE **Ovaries**,[1] two in number, project from the posterior lamina of the broad ligament, near to the sides of the pelvis. The diameter of each ovary is 1 inch, by $\frac{3}{4}$, by $\frac{3}{8}$ of an inch. The posterior surface looks backwards, the anterior being attached to the broad ligament. Their long axis is either perpendicular or somewhat transverse. The part of the ovary joining the broad ligament is called the hilum. The ovary lies just below the level of the brim, and in front of the sacro-iliac joint.

The ovary is covered on its outer aspect by columnar epithelium—the germ epithelium of Waldeyer.[2] At the hilum of the ovary this epithelium is continuous with the endothelium of the peritoneum forming the broad ligament. Below the germ epithelium is the so-called 'tunica albuginea,'[3] and on section the rest of the ovary consists of a cortical zone, in which the Graafian follicles occur, and a medullary vascular zone, continuous with the tissue of the broad ligament. Pflüger's 'egg tubes' as seen in the ovary of a rabbit on microscopical section, appear as involutions from the germinal epithelium downwards into the stroma of the ovary, and are developed somewhat after the manner of tubular glands from the germ epithelium itself (Quain).

The **Graafian follicles** near the surface of the ovary are small, the larger ones generally lying deeper, except one or two of the ripest ones, which may be near the surface.[1] Each Graafian follicle consists of a tunica fibrosa and a tunica propria, the membrana granulosa and the liquor folliculi; usually the membrana granulosa exhibits a projection of cells (discus proligerus), which contains the ovum.

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[1] Berry Hart, Allbutt and Playfair's *System of Gynæcology*, p. 49; 'Anatomy of Abdomen and Pelvis,' Cripps's *Ovariotomy and Abdominal Surgery*, chap. i. 1898: and Quain's *Anatomy*, vol. iii. part iv. p. 271.

[2] Quain, *ibid.* p. 274.

[3] 'Beneath the germ epithelium a firm layer of fibrous tissue encloses all the deeper parts. This has been compared to the dense fibrous covering of the testicle, and thence named "tunica albuginea," but without sufficient reason, for it is not a distinct tissue, and is, in fact, no more than a condensed part of the ovarian stroma' (Quain).

The **Ovum** consists of zona pellucida, yelk, germinal vesicle (nucleus), and germinal spot (nucleolus).

The **Broad Ligaments** consist of two folds of peritoneum passing out from the sides of the uterus to the side wall of the pelvis. The anterior fold of the broad ligament is a continuation of the peritoneum on the anterior surface of the uterus; beneath this lies the round ligament which passes from the junction of the Fallopian tube and uterus forwards and outwards to the inguinal canal; it is continuous with the musculature of the uterus and broad ligaments, and contains vessels and lymphatics. The posterior layer of the broad ligament is a prolongation backwards of the peritoneum of the posterior surface of the uterus; it passes much further downwards than the anterior, and lies partly on the side-wall of the pelvis. The ovary projects from this lamina.[2]

Between the layers of the broad ligament lie connective tissue, blood-vessels, lymphatics, and nerves; the connective tissue passing into that of the iliac fossa.

The **Ovarian Ligament** joins the lower end of the ovary at the angle between tube and uterus, and uterine muscle also passes into it. The Fallopian tube occupies the greater part of the top of the broad ligament, the free part of the broad ligament not occupied by the tube, and passing to the sides of the pelvis is called the 'infundibulo-pelvic' ligament. It contains the ovarian artery as it passes to the ovary.

The **Parovarium** [3] also lies between the layers of the broad ligament near the ampulla of the tube; it represents the remains of the Wolffian duct and body. Abnormal vestigial relics of the Wolffian body or of Müller's ducts (Kossmann) also occur at the hilum of the ovary and in the broad ligament.

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[1] 'The smallest Graafian follicles have no proper wall; they consist merely of a single layer of cells investing the contained ovum. In section the cells are flattened, and look spindle-shaped in the human subject, and not very unlike the cells of the ovarian stroma, so that they have been thought to be derived from those cells; but the observations of Balfour and others on the lower animals tend to confirm the view first taken by Waldeyer, that, like the ova themselves, the epithelium cells of the follicles are originally derived from the germinal epithelium' (Quain, vol. iii. pt. iv. p. 276).

[2] The portion of the broad ligament *above* the attachment of the ovary is the 'mesosalpinx'; that *below* should be called the 'mesometrium' (Waldeyer: *Das Becken*, 1898).

[3] Doran, *Diseases of Ovaries, Fallopian Tube, and Broad Ligament*, 1884, p. 40.

The parovarium proper consists of an outer series of tubes free at one extremity, called **Kobelt's Tubes**, and an inner set, the vertical tubules, of which generally 12 to 17 can be counted. These pass upwards into a larger tube, running at right angles to them, which may be occasionally traced to the vagina, known as **Gartner's Duct**,[1] corresponding to the vas deferens in the male. The tubes of the parovarium end blindly and are usually lined with epithelium.

The **Corpus Luteum.**—After rupture of a mature Graafian follicle, the cavity is first filled with blood, but after a time this shrinks, and is occupied by reddish yellow tissue undergoing fatty changes, seen often as a wavy, yellow band. The blood becomes gradually absorbed, the central space becomes obliterated, the yellow cells disappear, and finally only scar tissue and a few blood crystals remain. The term 'true' and 'false' corpus luteum should be abandoned.

Cystic Corpora Lutea.—These are small cystic bodies with thick walls, sometimes seen on the surface of an ovary, as large as a cherry, the walls of the cyst being of a bright yellow colour and containing an albuminous fluid. They cause no trouble, and probably do not give rise to ovarian cysts.

Corpora Fibrosa.[2]—These are due to some fibrous change in the corpus luteum, and are found as tough semi-opaque bodies in the ovary; their walls sometimes appear wavy in outline, and they may contain a cavity. The blood-vessels in the neighbourhood of these bodies are often thickened and degenerate as in 'arteritis obliterans.' Sometimes these fibrous bodies occur on the surface of the ovary and are pedunculated, or they may be calcified.

Calcified Corpora Lutea also occur embedded in the ovary; they are irregular and nodulated in shape, yellow on section, and consist of tough fibrous tissue impregnated with lime salts.

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[1] Bland-Sutton, *Surgical Diseases of the Ovaries and Fallopian Tubes*, 2nd edit. p. 90; Routh, *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 152.

[2] Patenko, 'Ueber die Entwickelung der Corpora Fibrosa in Ovarien' *Virchow's Archiv*, vol. lxxxiv., 1881. See also Bland-Sutton, *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 223.

Ovarian Concretions.[1]—These sometimes occur in the ovary. Bland-Sutton is of the opinion that they consist of coagulated proteids derived from blood-clot akin to lardacein, and analogous to the concretions of the prostate, and the amyloid bodies sometimes found in old hæmorrhages.[2] Doran[3] describes them as solid, claret-coloured bodies, sometimes lens-shaped and about a quarter of an inch in diameter, varying from a pea to a bean in size, generally with a smooth surface. They are very rare. They occur in ovarian follicles, and may be due to curious colloid[4] changes in the effused blood.

Apoplexy of the Ovary.[5]—This may be also termed hæmorrhage into the ovary, or hæmatoma, and in it through rupture of a follicle, bleeding takes place into the stroma of the ovary, converting the organ into a cyst, the walls of which are formed of expanded ovarian tissue.

This must not be confounded with mere follicular hæmorrhage, which occurs into a large mature Graafian follicle, and which may be of some size, nor with hæmorrhage into an ovarian cyst, which occurs after tapping, twisting of the pedicle, or rupture of vascular adenomatous growths in their interior. Hæmorrhage into the ovary is gradually absorbed, and the clot decolorised, only fibrin and scar tissue remaining. It may be the cause of great pain, and the increased size of the ovary may lead to prolapse. The cause is not known.[6]

Age Changes in the Ovary.[7]—In the fœtus the ovary is an elongated body lying parallel with the Fallopian tube, with wavy borders, and with a longitudinal furrow on its free surface; at puberty it becomes larger, rounder, and firmer, and during active sexual life remains much the same, but its surface is pitted and scarred from the effects of repeated rupture of ripe Graafian follicles; the size varies in different individuals.

After the climacteric, the ovary begins to shrink, its surface becomes irregular and wrinkled, and at the same time the ova and ovarian follicles gradually disappear,[8] until, in very advanced life, nothing is left but a corrugated body consisting of fibrous tissue traversed by a few blood-vessels and weighing about 15 grains, whereas an active ovary weighs on an average 90 grains.[1] The uterus and Fallopian tubes undergo corresponding atrophic changes.

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[1] Bland-Sutton, *Surgical Diseases of the Ovaries and Fallopian Tubes*, 1891, p. 21.

[2] Hector Mackenzie, *Trans. Path. Soc.* vol. xl. 1889, p. 198.

[3] Doran, 'Blood Concretions of the Ovary,' *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 214.

[4] Bland-Sutton, *op. cit.*

[5] Doran, *Trans. Obstet. Soc. Lond.* vol. xxxii. 1890, p. 119.

[6] 'It is possible that some of these curious hæmorrhages into the ovary are due to Primary Ovarian Pregnancywith the formation of a mole.'—AUTHOR : see p. 153.

[7] Bland-Sutton and Giles, p. 250.

[8] Bland-Sutton, *op. cit.* new ed. 1896, p. 27.

These age changes in the ovary are, however, by no means constant, and the appearances of a healthy fertile ovary may vary between wide limits. Many women with small puckered ovaries become repeatedly pregnant, and others with large apparently healthy ovaries are sterile, and sterility may be accounted for in other ways. Many such ovaries are described as pathological, when the condition is really physiological. Naked-eye appearances of supposed 'cirrhosis' must not be trusted, and microscopical evidence is not always trustworthy. Even changes described as inflammatory, and indicative of oöphoritis, may possibly be physiological. No one knows how much ovarian tissue is necessary for ovulation, for conception occurs in some women even when both ovaries are most extensively diseased.

Abnormalities of the Ovary.[2]—Congenital absence of both ovaries is very rare, and supernumerary ovaries do not occur. Bodies so described are probably partially detached tubes of the parovarium, stalked corpora fibrosa, or small myomata of the ovarian ligament. Absence of one ovary usually accompanies deficient development of the corresponding half of the uterus and Fallopian tube, and absence or misplacement of the corresponding kidney. With uterus unicornis the ovary often retains its infantile shape.[3]

Atrophy of the ovary[4] may occur with deficient uterine development in the insane; in large tumours in the abdomen it may be atrophied from pressure, or follow upon severe pelvic inflammation and suppuration.

Hernia of the Ovary.—This may be either acquired or congenital. In the latter case it is associated with persistence of the processus vaginalis of Nuck; when acquired, it is generally inguinal, less frequently femoral, but the ovary has been found in ventral, gluteal, obturator, and umbilical herniæ.

Congenital hernia of the ovary is, it appears, exceedingly rare,

[1] 1 gramme = 15·432 grains; 1 oz. = 28·35 grammes.

[2] Bland-Sutton and Giles, *Diseases of Women*, 1st ed. p. 251.

[3] Bland-Sutton, *op. cit.* 1891, p. 32.

[4] Doran, *Trans. Obstet. Soc. Lond.* vol. xxi. 1879, p. 253.

but hernia of the ovary may occur in infancy, or at any period of life. Many herniæ of the ovary formerly described as such are really hydroceles of the canal of Nuck,[1] and swellings described as ovaries in hermaphrodites must be carefully examined before they are pronounced to be ovaries. Bodies so described have turned out to be testes,[2] even when a uterus and well developed external genitals existed. Hernia of the ovary may be accompanied by the Fallopian tube, or, more rarely, by the uterus or one horn of a double uterus, or intestine, to which the ovary is adherent. Frequently a herniated ovary is cystic or diseased. All supposed hernial ovaries on removal should be subjected to microscopic examination.

Prolapse of the Ovary.—This may be caused by pregnancy, retroflexion of the uterus, or enlargement of the ovary itself. Such descent may be sudden, but more often it is gradual, and it must be remembered that the uterus and pelvic floor are often in a similar stage of descent. Tenderness[3] of the prolapsed ovaries is the chief symptom, perhaps the only one, and it is all the more serious if such ovaries become fixed by adhesion; one of the factors of such prolapse is the relaxation and stretching of the pelvic peritoneum and floor, due to repeated pregnancy, and the condition called subinvolution. Much the same applies to retroflexion where the ovaries fall backwards with the uterus, and care should be taken after any confinement to avoid this condition, indeed to avoid anything which may lead to delay in normal involution. When an ovary is enlarged it tends by its own weight to fall low in the pelvis; œdema, inflammation, tubercle, apoplexy, and cystic disease may be the cause of such enlargement. It is very rare to get an undescended ovary.[4] In such cases the ovary may remain near the kidney, or in some position between the kidney and the brim of the pelvis; if so it may retain its infantile shape, and the cæcum may also fail to descend into the right iliac fossa.

Inflammation of the Ovary.—Inflammation of the ovaries, 'oöphoritis' as it is called, meaning by the term inflammatory changes so marked that they are undoubted, is always associated with pelvic peritonitis or cellulitis, and often with inflammation of the tubes, and is included best under the term

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[1] Bland-Sutton, *Surgical Diseases of the Ovaries &c.*, 1891, p. 28.

[2] Kossmann in Martin's *Krankheiten der Ovarien*, 1898, p. 158.

[3] Griffith, *op. cit.* p. 869.

[4] Martin, *loc. cit.* p. 146.

'inflammation of the uterine appendages'; for practically all forms of oöphoritis such as this are accompanied with 'fixation and other evidences of surrounding inflammation,' and it is very doubtful if oöphoritis as described in text-books unaccompanied by fixation occurs, or, at all events, if it can be recognised; and even if the microscope be employed it must not be forgotten that the appearances found in an ovary may not be pathological at all, or they are assumed to be pathological in order to explain the causes for which such an ovary was removed. 'Cirrhosis of the ovary'[1] is 'a name denoting an assumed disease which we know nothing about, but which is the excuse for the operation. No signs, microscopic or macroscropic, have yet been described by which, if two ovaries of about the same size, and not showing signs of adhesions, are laid together on a plate, an ovary which was painful can be distinguished from one which was not.'

Oöphoritis as a real disease apart from pelvic inflammation probably does not occur, and will not be described.

With pelvic inflammation (perimetritis) two kinds can occur:

a. Peri-oöphoritis.
b. Abscess of the ovary.

Peri-oöphoritis—that is, inflammation of the peritoneum around the ovary, resulting in adhesions to adjacent parts—is but a part of perimetritis, and such an ovary is bound down and adherent to tube, uterus, or bowel. Such changes are said to be accompanied by thickening and induration of the surface of the ovary, and to interfere with, or prevent the rupture of, Graafian follicles; and, further, such peri-oöphoritis may extend into the substance of the ovary, producing induration and changes in the stroma, leading to degeneration of the follicles. Peri-arteritis of vessels is also described, and even morbid cystic changes. It is very doubtful if such can be accepted as definitely due to the peri-oöphoritis, or even if they are morbid changes at all; and before we can interpret them as pathological, an accurate knowledge[2] of the normal ovarian structure at different periods of adult life must be obtained. We have no evidence that such chronic inflammation of the ovary as described above adds to the symptoms, or modifies the course, of the perimetritis on which it depends.[1]

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[1] Herman, *Diseases of Women*, p. 73.

[2] Griffith, Allbutt and Playfair, *op. cit.* p. 862.

Abscess of the Ovary.—The most acute forms of inflammation of the ovaries are those resulting from septic infection in connection with parturition, abortion, instrumentation, and with gonorrhœa. In fatal cases, such an ovary is found enlarged, soft, and sloughing; extravasations of blood and pus may be seen on section in the stroma and follicles, while the uterus, tubes, and cellular tissue of the broad ligament will be found in a similar condition.[2] Such minute foci of suppuration in the ovary may extend, coalesce, and form an abscess in the ovary [3] of considerable size, sometimes with very thick walls, and possibly long quiescent; it may quiet down, or be a cause of serious danger. Abscess of the ovary is one of the most frequent complications of purulent salpingitis,[4] the ovary becoming apparently directly infected from the diseased tube, or it may spread along the broad ligament as a cellulitis.

A variety of inflammation described by Olshausen[5] is called 'oöphoritis serosa,' or what is mostly described as œdema of the ovary. It is said to be produced by gonorrhœa and pelvic inflammation, or to follow fevers and mumps. Such ovaries are enlarged, swollen, smooth, and œdematous, with no folds or scars. They are very heavy, and are likely to become prolapsed. Whether oöphoritis serosa is a mere œdema or an inflammation it is difficult to say.

The writer has described a very similar condition in the ovaries, found with uterine fibroids, which is certainly due to back-pressure and œdema, and not to inflammatory changes at all.[6]

Tubercle of the Ovary.[7]—This is a very rare condition, and is generally part of a general tubercular condition, and therefore secondary. It occurs in the ovary:

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[1] Herman, *op. cit.* p. 219.

[2] Lewers, *Trans. Obstet. Soc. Lond.* vol. xxx. 1888, p. 7; Targett, *ibid.* vol. xxxvii. 1895, p. 216.

[3] Griffith, Allbutt and Playfair, *op. cit.* p. 863.

[4] Cullingworth, *Lancet*, July 2 and 9, 1892; *id. Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 277; *id. Clin. Illustrations of Diseases of Fallopian Tubes and Ovaries*, p. 16, plate v.

[5] Olshausen, *Die Krankheiten der Ovarien*, 1886, and Griffith, *op. cit.* p. 864.

[6] See chapter on 'Uterine Fibroids,' p. 173.

[7] Griffith, *Trans. Path. Soc. Lond.* vol. xl. 1898, p. 212; Whitridge Williams, *Johns Hopkins Hospital Reports*, vol. iii. 1893; and *Trans. Amer. Gynæc. Soc.* vol. xvi.

- *a*. As miliary tubercle of the surface, usually associated with tubercle of the peritoneum.
- *b*. Tubercle in the substance of the ovary, leading to caseation and abscess.

In the first class of case the ovary may or may not be enlarged, and the diagnosis is only made by inspection. In the second, where abscess forms, it does not differ clinically from ordinary abscess of the ovary.

The disease may be suspected in a tubercular subject where the ovary is enlarged, but before removal its distinct tubercular characters and the caseous masses in the abscess cavity will not be evident, and there is nothing peculiar in the symptoms of perimetritis or salpingitis caused by tubercle. A tuberculous abscess of the ovary sometimes causes death by bursting into the peritoneal cavity, or may form fistulous communication with the rectum. Bland-Sutton [1] says that an ovarian abscess unassociated with salpingitis is in nearly all cases tubercular.

Tumours of the Ovary.—Nothing, perhaps, is more puzzling than the investigation of neoplasms arising from the ovary, and a classification on any reasonable lines seems almost impossible, for 'while the characteristic features of a principal class are readily recognisable, the variations of them are so numerous that, in the present state of our knowledge, it is often not practicable to classify a particular tumour with certainty, for innocent tumours pass by almost insensible gradations into malignant, solid tumours, develop cysts, and, *vice versa*, papillomatous growths develop both in cystic tumours and on the surface of the ovary without any cystic formation, and cysts with adenomatous, papillomatous, or dermoid contents occur either alone or as parts of tumours of different kinds.' [2]

With regard to their *Etiology*, we are still very much in the dark, for ovarian tumours include not only tumours growing from the ovary itself but from other regions in the neighbourhood, such as the hilum and parovarium, and a knowledge of these parts and their structure in health is necessary before any pathological inferences can be drawn as to the origin of their neoplasms.

The Graafian follicle has already been described, and it seems reasonable to believe that many ovarian tumours arise in this way[1]; and Bland-Sutton[2] has pointed out that, morphologically at least, the Graafian follicle is a modified mucous gland, and lined with cells (the membrana granulosa) derived really from the genital ridge of the cœlom, and therefore in a way comparable to those lining the intestine. In the follicle of the cat[3] these cells are tall and columnar, in no way differing from the cells lining the spaces of ovarian adenomata.

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[1] Bland-Sutton, *Surgical Diseases of the Ovaries &c.*, 1891, p. 276.

[2] Griffith, Allbutt and Playfair, *op. cit.* p. 836.

Whether or not we can regard the ovary itself as a mucous gland, and further a ductless gland, with a secretion, like the thyroid, cannot at present be determined. Ovarian secretion has not yet been proved, but the resemblance of tumours of the ovary and thyroid, and the extraordinary results of removal of the ovaries on the general nervous system, make this subject worthy of consideration, though it is not perhaps capable of proof. Certainly, however, the lining membrane of many of the glandular adenomatous cysts must be regarded in the light of a mucous membrane,[4] and in the so-called colloid cysts the most wonderful tall columnar epithelium with typical goblet cells can be seen (fig. 93); another point of importance is that there is very little difference between skin and mucous membrane, except that of function, and it is possible that the Graafian follicle[5] can in itself provide all the developmental requirements even for such curious tumours as Dermoids. ‘Inclusion of epiblast’ or ‘parthenogenesis’ are mere words and settle nothing.[6]

Turning to the connective tissue of the ovary, no doubt many solid tumours arise in this way, especially the sarcomata,[7] while in the region of the hilum (par-oöphoron) and from the parovarium we have a series of developmental peculiarities in the remnants of the Wolffian body, which without much doubt are the origin of some of the papillomatous cysts arising in that position, and which do not necessarily involve the ovary at all.

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[1] Wilson Fox, ‘Origin of Ovarian Tumours,’ *Trans. Med.-Chir. Soc.* vol. xlvii.

[2] Bland-Sutton, *Tumours, Innocent and Malignant*, p. 344.

[3] Bland-Sutton, *Surgical Diseases of Ovaries &c.*, 2nd ed. 1896, p. 3.

[4] Bland-Sutton, *op. cit.* 1891, p. 49; and Poupinel, *Arch. de Physiologie*, ser. iii. vol. ix.

[5] Doran, *Tumours of Ovary, Fallopian Tube, and Broad Ligament*, 1884, p. 78.

[6] Bland-Sutton, ‘Dermoids,’ in *Tumours, Innocent and Malignant*, p. 341.

[7] Foulis, ‘Malignant Ovarian Tumours,’ *Brit. Med. Journ.* 1878, vol. ii. pp. 91, 658.

Cystic and other degenerations of the corpora lutea have already been described; they probably do not give rise to any tumour of importance.[1]

Noeggerath, Rosthorn, Leopold and Mary Dixon Jones[2] have described tumours arising from the blood-vessels of the ovary or from walls of lymphatics; Waldeyer,[3] Schroeder, and Spiegelberg believe that tumours may arise from true Pflüger's tubes, while de Sinéty and Malassez[4] think that certain epithelial tubes in the ovary, probably not true Pflüger's ducts, may be the origin of other tumours.

Balfour, Foulis, and Doran[5] deny that Pflüger's tubes exist, as such, in the healthy mammalian ovary, and Doran[6] says that he 'cannot help thinking that the origin of cystic disease is to be sought from careful and prolonged study of the different changes which follicles in process of atrophy may undergo, when influences which it may be impossible to trace prevent the atrophy from ever being completed.' Probably the view that the Graafian follicle is the origin of a large number of ovarian tumours has most acceptance among English pathologists and embryologists.

Classification of Ovarian Tumours.—This, as previously pointed out, seems almost impossible, and only the chief types will be mentioned. Doran subdivides tumours according to their initial origin from the periphery, the hilum of the ovary, and from the broad ligament; this is more or less followed by Bland-Sutton in the distinction of oöphoritic cysts, paroöphoritic (hilum) cysts, and parovarian cysts.

Clinically, at all events, we can recognise the following classes of tumours, which will be shortly described.

Varieties of Ovarian Tumours.—These may be broadly classified (*a*) **according to their position** into :—

1. Tumours arising from the periphery of the ovary, which contains Graafian follicles (oöphoron).

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[1] H. Kelly, *Operative Gynæcology*, vol. ii. p. 180.

[2] Noegerrath, *Amer. Journ. of Obstetrics*, vol. xii. 1880; Dixon Jones, *New York Med. Journ.* 1890; Leopold, *Arch. für Gynäk.* vol. vi. p. 159.

[3] Waldeyer, *Arch. für Gynäk.* vol. i. p. 252.

[4] Malassez and de Sinéty, *Arch. de Physiol. et Pathol.* 1878, p. 343; and Griffith, Allbutt and Playfair, p. 843.

[5] Doran, *Tumours of Ovary, Fallopian Tube, and Broad Ligament*, pp. 2 to 5.

[6] Doran, *op. cit.* p. 7, 'Arrest of the Normal Retrograde Metamorphosis of Graafian Follicles,' p. 12.

2. Tumours arising from the hilum of the ovary, which contains Wolffian relics (paroöphoron).
3. Tumours arising either from the parovarium proper, or from other portions of the broad ligament.
4. Tubo-ovarian cysts (so called).

or (*b*) **according to their structure :**

- 1. Cysts
  - *A.* Simple unilocular.
  - *B.* Proliferous.
    - 1. Simple.
    - 2. Adenomatous.
    - 3. Papillary.
    - 4. Malignant.
  - *C.* Dermoid.
- 2. Solid Tumours
  - *A.* Fibroma.
  - *B.* Myoma.
  - *C.* Sarcoma.
  - *D.* Carcinoma.

Simple Unilocular Cysts.—Probably a true ovarian cyst is seldom really unilocular, and the term is only of clinical significance. Generally, somewhere in the wall of the main cyst cavity, some other loculi can be found, or bands and septa remain, indicating its original compound character. Small unilocular cysts are sometimes lined with epithelium identical with the membrana granulosa,[1] proving such a cavity to have been an ovarian follicle, and the cyst can be clearly seen occupying the periphery of the ovary, covered with the original germ epithelium, not having invaded the hilum at all. Larger cysts lose their original relations, and their cavities may be lined with flat epithelium, or there may be no epithelium at all, owing to atrophy by pressure of the contained fluid. Sometimes the lining membrane is soft, or there may be raised patches or elevations which are exactly like mucous membrane in complex folds. Such cysts are often quite small and unimportant; they are generally found accidentally.

Simple Hydrops Folliculi may occur but it is difficult to say when such a distended follicle should be called a cyst. It is possible that small single cysts of ovary may arise in this way; they are lined with cells like the membrana granulosa, and contain a mucoid fluid; often the remainder of the ovarian tissue can still be demonstrated. Cysts of the corpora lutea already

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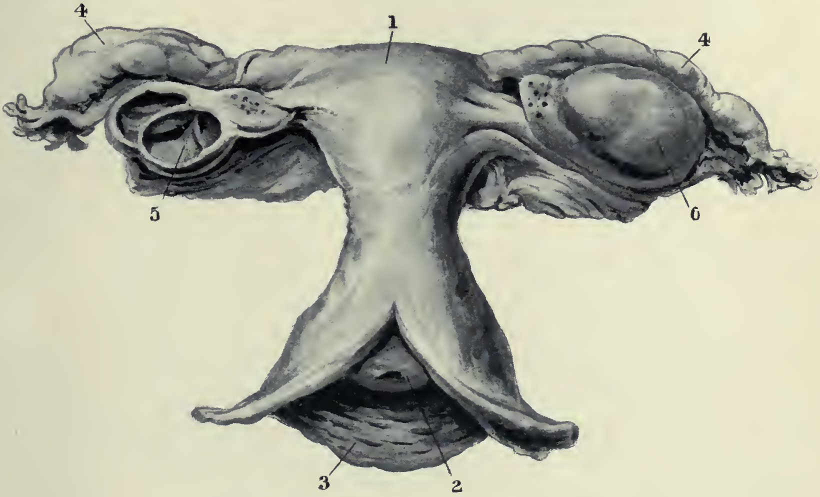
[1] Bland-Sutton, *Surgical Diseases of the Ovaries &c.*, 1891, p. 46.

FIG. 83.

CYSTIC OVARIES IN A CHILD. (A LITTLE LARGER THAN NATURAL SIZE.)



1. Fundus uteri. Note its small size in comparison with the cervix.
2. External os uteri.
3. Vagina.

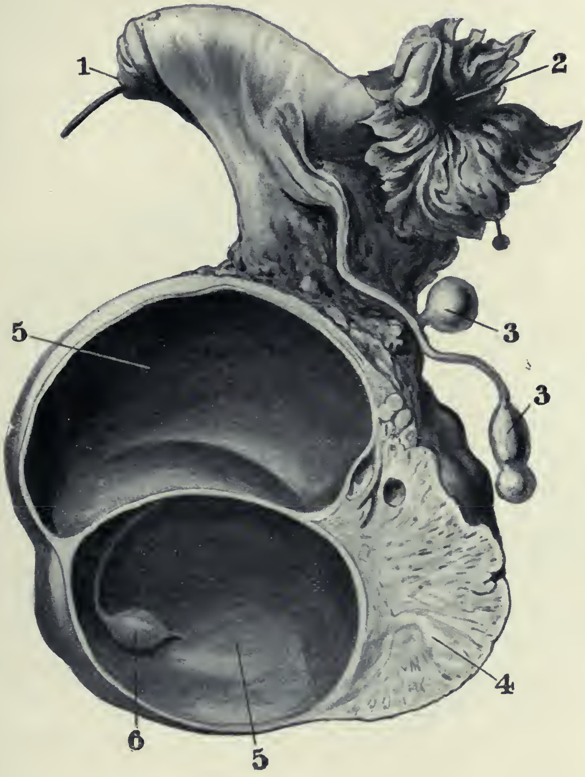
4, 4. Fallopian tubes.

5. Ovary of the left side, a portion of which has undergone cystic changes.
6. The opposite ovary, the outer and upper portion of which is occupied by a cyst. The elongated form of the ovary in infancy is well shown in the above specimen.

(Specimen 2348. Museum, St. Thomas's Hospital.)

FIG. 84.

EARLY CYSTIC DISEASE OF THE OVARY.



An ovary, Fallopian tube, and portion of the broad ligament, to show early cystic disease of the ovary itself. A considerable portion of normal ovarian tissue still remains.

1. Uterine end of Fallopian tube.
2. Fimbriated extremity. The fimbriæ are very luxuriant; a small pedunculated cyst is seen growing from one of them.

3, 3. Curious stalked cysts, with a long pedicle growing from the region of the parovarium; they belong to the broad ligament. (Kobelt's tubes?)

4. Normal ovarian tissue.

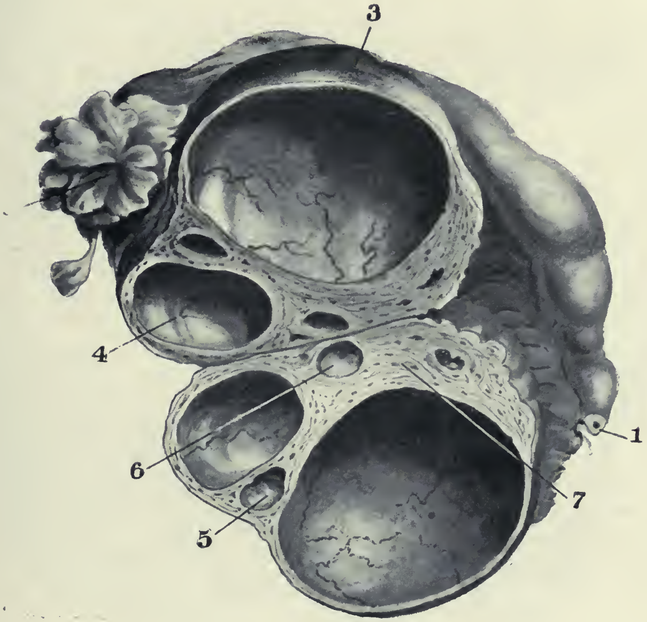
5, 5. Two simple cysts of the ovary, probably arising from Graafian follicles

6. A small secondary cyst.

(Specimen 2349. Museum, St. Thomas's Hospital.)

Fig. 85.

EARLY CYSTIC DISEASE OF THE OVARY. (Natural Size.)



A Fallopian tube, with an ovary in section showing early cystic degeneration. Portions of normal ovarian tissue still remain. The cysts occupy the periphery of the ovary, and probably arise from altered Graafian follicles.

1. Uterine end of Fallopian tube.
2. Fimbriated extremity.
3. Large simple cyst near surface of the ovary.

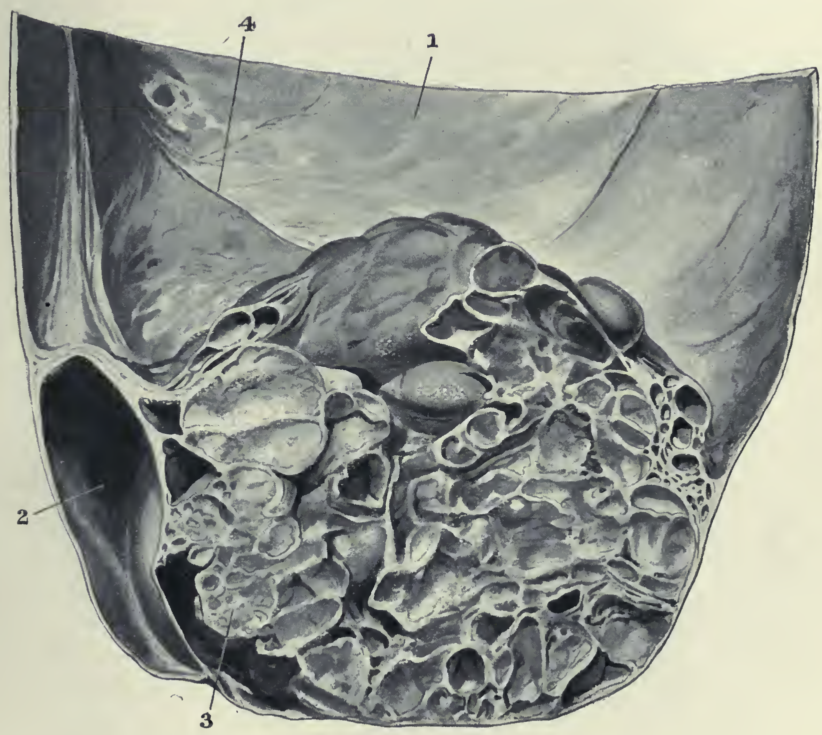
4, 5, 6. Smaller secondary cysts.

7. Remains of normal ovarian stroma.

(Specimen 2904 G. Museum, St. Bartholomew's Hospital.)

Fig. 86.

PORTION OF A LARGE MULTILOCULAR (GLANDULAR) OVARIAN CYST.



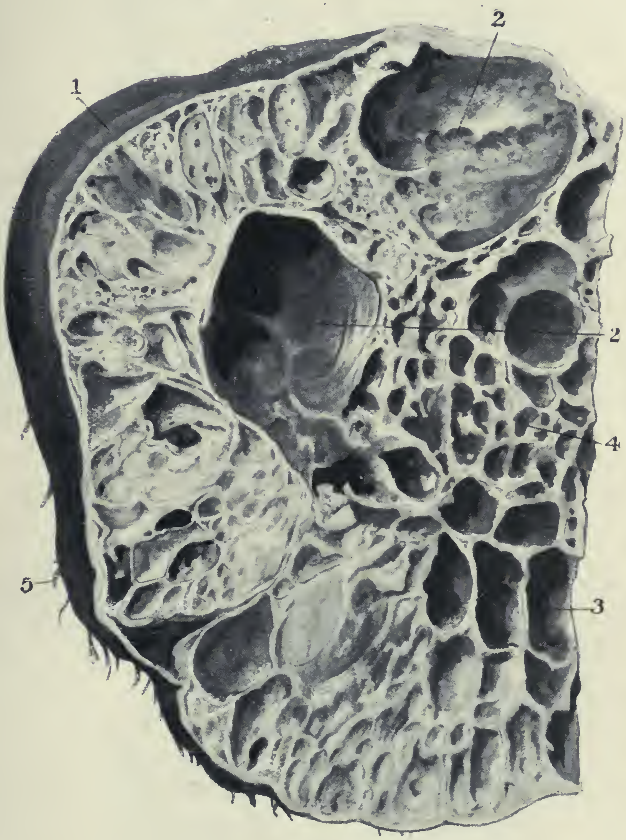
Section of a portion of a large ovarian cyst, showing the wall of the main cyst, and at its lower border a mass of smaller secondary cysts of great complexity. The smaller cysts contained clear thick mucoid material, and are lined with columnar (glandular) epithelium. The tumour is a simple cystic adenoma of the ovary.

1. Main cyst wall.
2. Secondary cyst cavity.
3. Mass of smaller honeycomb cysts contained within the main cyst wall.
4. Septum, showing remains of a cyst which has opened into the main cyst cavity.

(Specimen 2356. Museum, St. Thomas's Hospital.)

Fig. 87.

PORTION OF A MULTILOCULAR OVARIAN CYST (CYSTIC ADENOMA).



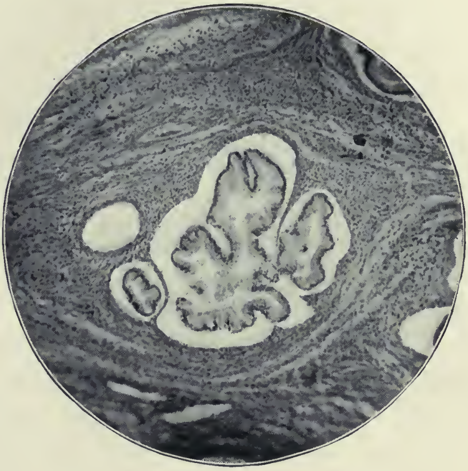
Portion of an ovarian cyst (which weighed 25 lbs.), showing the multilocular formation of an ordinary cystic adenoma of the ovary. In some parts the secondary cysts have the appearance of a honeycomb. The cysts are full of mucin.

1. Outer surface of main cyst wall.
2, 2. Large secondary cysts.
3, 4. Smaller cysts.
5. Adhesions on the surface of the main cyst.

(Specimen 3094 k. Museum, St. Bartholomew's Hospital.)

FIG. 88.

EARLY STAGE OF OVARIAN CYST FORMATION.

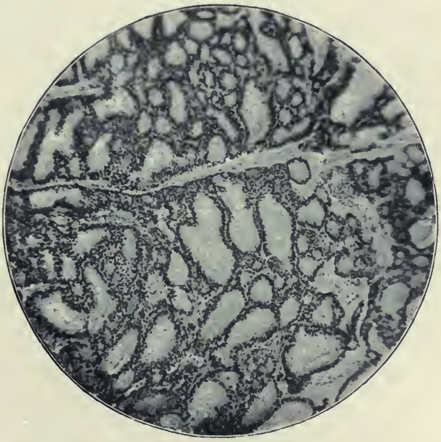


Section of an ovary, showing the early stages of cystic degeneration. Several irregular spaces are seen lined with tall columnar epithelium like that of a mucous gland. The cells have shrunk away from the wall of the cyst during the processes of hardening; in one space to the left they have dropped out entirely. The lumen of the spaces contains mucin.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

FIG. 89.

SIMPLE ADENOMATOUS OVARIAN CYST.



Section of the more solid portion of a simple multilocular glandular cyst of the ovary under low power, to show the complex nature of the growth. Innumerable cystic spaces are seen in the section, lined by a simple layer of columnar epithelium like that of a mucous gland. In some areas the spaces are large and filled with mucin; in others the spaces are smaller and crowded together in great complexity. The growth is a simple adenoma.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

described may occur in the same way; they are lined with yellow material at their periphery, like that of the corpus luteum. They are unimportant.

Proliferous Cysts.—These include

a. Simple multilocular cysts.
b. Cysts with adenomatous growths in the secondary cysts.
c. So-called papillomatous cysts.
d. Cysts in which either the cells lining the secondary cysts, or the stroma, have taken on a maligant type.

Simple Multilocular Cysts. (Figs, 83, 84, 85.)—In these cysts there has been distension in many of the follicles, so that a tumour forms composed of little cysts closely packed together. The simplest form is said to occur in Rokitansky's tumour,[1] a mere name given to ovaries slightly enlarged from multiple cyst formation; it is rare, and further it is not at all clear that this condition is not an early stage of the ordinary multilocular cyst.

Multilocular Cysts with Adenomatous Growth (Multilocular Adenomata or Glandular Multilocular Cysts).—These cysts probably in most cases arise from the periphery of the ovary (oöphoron), and are the most common form of ovarian tumour. They consist of a large cyst itself composed of and enclosing innumerable other cysts (fig. 86) of all sorts and sizes, some bulging the parent cyst-wall, others projecting into its cavity; sometimes such a cyst has few secondary cysts within it, or it shows evidence in its interior of septa (figs. 23, 86), the remains of cysts which have opened into one another. Other cysts are almost solid, with secondary cyst formation, and on section innumerable honeycomb masses of delicate translucent secondary cysts (figs. 86, 87) are seen filled with thick viscid fluid; such cysts are lined with columnar epithelium identical with mucous membrane (fig. 88), the larger cavities having a more flattened epithelium, while in the main cyst cavity only a few pavement epithelial cells may remain, altered by the pressure of the contained fluid. The main cyst-wall itself consists of tough fibrous tissue.

Sections of such tumours show structures of great complexity, chiefly that of innumerable cyst proliferation (fig. 89), probably by repeated budding of the original cysts, again forming secondary cysts, and so on ; these being further crowded together, by pressure, into hopeless complexity (figs. 90, 91). At the same time invagination of the cyst-wall by the surrounding stroma takes place, akin to that of cystic adenoma of the breast, but it is not really an intracystic growth as such.[1]

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[1] Herman, *Diseases of Women*, p. 727.

In some cysts of this nature there is not only a simple lining of columnar epithelium, but such epithelium itself may proliferate and grow into the cyst itself, at first as tiny processes, which, later, branch like the villi of the Fallopian tube and exactly resemble a mucous membrane as pointed out by Poupinel and Bland-Sutton.[2] These processes are to all intents and purposes adenomatous ingrowths, and cysts containing them may be designated cystic adenomata. They appear to be a stage between multilocular ovarian tumours composed simply of cysts, and the papillomatous tumours to be described presently (figs. 91, 92).

Certainly, if such ingrowths start from the lining membrane of the cyst-wall, and such membrane partakes of the glandular type, these growths should be termed adenomata, and the writer is of the opinion that all intracystic growths are of this nature primarily, and that even in the so-called papillary cysts the intra cystic growth is still adenomatous in origin.

Size and Rate of Growth.[3]—Multilocular adenomatous cysts sometimes reach enormous proportions, but not so frequently as they used to, before the days of ovariotomy. Cysts have been removed weighing with their fluid 150 lbs. ; in St. Thomas's Hospital Museum is a dried cyst which weighed 160 lbs., removed by Cullingworth.

The tendency of all ovarian tumours is to grow larger. They first displace the uterus and the pelvic contents, or the uterus

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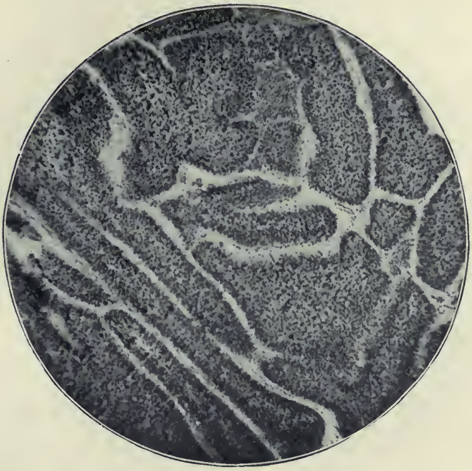
[1] Doran, *Tumours of Fallopian Tube, Ovary, and Broad Ligament*, 1884, p. 33.

[2] Poupinel, *Arch. de Physiologie*, ser. iii. vol. ix. ; and Bland-Sutton, *Surgical Diseases of Ovaries &c.*, 1891, p. 48.

[3] Bland-Sutton, *op. cit.*, 2nd ed. pp. 80–82. Cyst removed by Dr. Elizabeth Reifsruder (*Amer. Journ. Obs.* vol. xxxi. p. 512), = twenty-two gallons of fluid ; cyst and contents weighed over 226 lbs. H. Kelly, *Operative Gynæcology*, vol. ii. p. 253. Bullitt, *Annals of Gynæcology* (America), November 18, 1899 : report of a cyst weighing 245 lbs. removed by Dr. Cartledge, of Louisville, with records of twenty-three cases in which the tumour weighed over 100 lbs. (*Brit. Gyn. Journ.* January and February, 1900). F. A. Baldwin, *Brit. Med. Journ.* vol. 11, 1900, p. 79 ; tumour 185½ lbs. ; fluid 18 gallons. For rate of growth, see also Bland-Sutton, *op. cit.*, 2nd ed. p. 83.

Fig. 90.

SOLID PORTION OF AN OVARIAN ADENOMA.

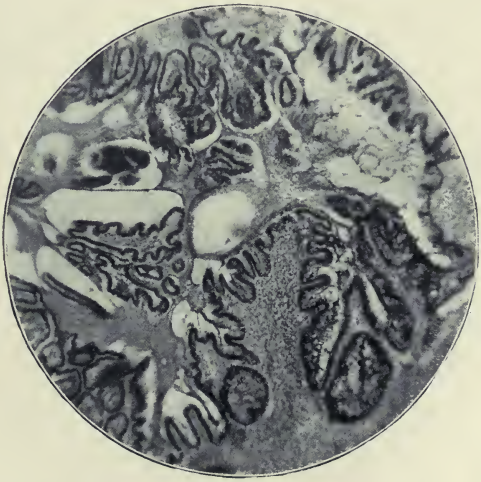


Section of the solid portion of an ordinary multilocular glandular cyst of the ovary. The specimen exhibits the later complex changes of the growth. Irregular spaces are seen crowded to their fullest extent with degenerate cells, the result of proliferation of the columnar epithelium which originally lined them; owing also to pressure, the acini and the cells lining them have become crowded together, especially to the left of the section. The growth is innocent.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 91.

SOLID PORTION OF AN OVARIAN CYST.

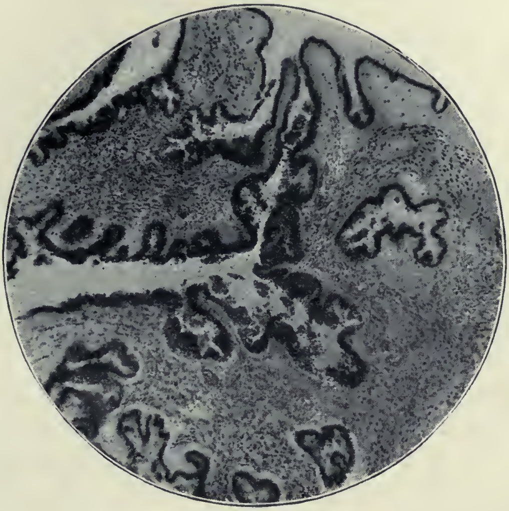


Section of the solid portion of a large adenomatous ovarian cyst. It shows the complicated multilocular formation of the growth. Everywhere are seen irregular acini of great complexity lined with columnar epithelium like that of a mucous gland. The stroma is dense, and consists mostly of fibrous tissue, while the mass of the tumour consists of adenomatous material. It is not malignant.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 92.

SECTION OF THE WALL OF AN OVARIAN CYST (ADENO-PAPILLOMATOUS).



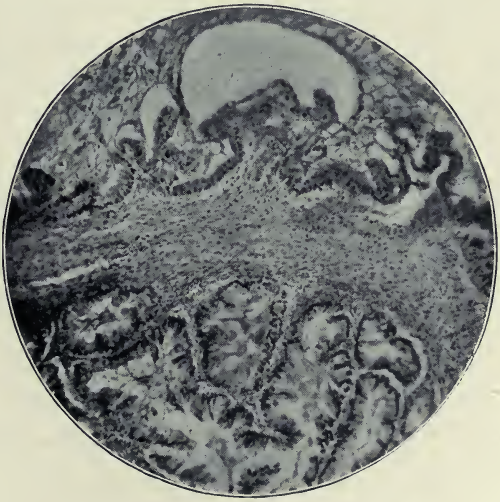
Section of portion of an ovarian cyst. In the lower part of the section typical spaces are seen lined with columnar glandular epithelium. The spaces are very complex and irregular towards the centre of the section.

At the upper part, which projected into the cyst cavity, the stroma is harder and thrown into papillæ covered with a single layer of epithelium. Adenomatous and papillomatous changes therefore are present in the same section.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 93.

COLLOID DEGENERATION OF AN OVARIAN CYST.



Section of part of the wall of an ovarian cyst which was filled with thick apple-jelly coloured material. In the lower part of the section are acini containing colloid material lined with the most beautiful tall columnar epithelium. These acini are identical in structure with a mucous gland; many of the cells are goblet cells.

In the upper part of the section the cells are degenerate, and in parts the colloid material is seen amid the stroma.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

itself may be adherent to the tumour or displaced by the traction on the pedicle. The bladder and rectum may be displaced by large tumours and the intestines crowded up under the ribs. Fixed tumours may interfere with important pelvic structures. Simple cysts grow with far greater rapidity than adenomata or dermoids.

The Fluid in Glandular Cysts.—This is generally of fairly high specific gravity, 1010 to 1030; it contains much albumin and mucin. The 'ovarian cells' in such fluid have long since lost their significance.

The albumins are complicated, consisting of paralbumin and metalbumin. The former is closely allied to mucin, and the latter Hammarsten[1] designates as 'pseudomucin.' Pfannenstiel[2] calls the same substance 'glyco-proteid.'

This pseudomucin is very abundant in cysts containing the tough sticky fluid; it is a secretion, not a degeneration. It is to be noticed that the fluid in the smallest cysts is generally thicker than in the larger cavities. Pseudomucin does not occur in parovarian cysts, and rarely in papillary ones. In other glandular tumours the fluid is so thick that it will not run out at all through a trocar, and is so tenacious that it has to be drawn out by the hands in long viscid masses, or it may be like boiled starch, yellowish, even orange-coloured; it is particularly so in cysts undergoing colloid degenerative changes.[3] In microscopical sections, colloid cysts exhibit the most remarkable tall columnar goblet cells (fig. 93) lining some of the secondary cyst cavities; in other areas the epithelial cells themselves have undergone colloid (mucoid ?) changes, while large areas of deeply staining gelatinous masses appear sometimes to penetrate into the stroma in long irregular masses containing only a few remains of altered cells, and in some places the colloid material appears to be laminated.[4]

Cholesterine and earthy salts occur frequently in old glandular cysts.

The gritty particles sometimes found amid the more solid portions of such cysts are called psammomata; they also occur in papillary cysts, and are probably due to degenerative processes.

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[1] Hammarsten, *Zeits. f. Phys. Chem.* 1882.

[2] Pfannenstiel, *Arch. f. Gyn.* vol. xxxviii. 1890, p. 407; also H. Kelly, *Operative Gynæcology*, vol. ii. p. 262.

[3] Mary Dixon Jones (New York), 'Colloid Degeneration of the Ovary,' *Brit. Gyn. Journ.* January and February, 1900.

[4] Sims Woodhead, *Practical Pathology*, 3rd ed. p. 606.

Papillary Cysts (so called) (figs. 94, 95, 96).—Whether these tumours can be definitely described apart from ovarian adenomata is disputed, as already pointed out. Some histologists hold that there is no essential difference as to their place of origin, and the occurrence of mixed forms of tumours favours this view. The general opinion, however, in this country is that stated by Bland-Sutton and Doran.[1] The latter says that the multilocular cysts of a glandular nature arise from the stroma of the parenchyma (oöphoron), while from the tissue of the hilum (paroöphoron) arise the multilocular proliferous cysts which contain firm exuberant papillomatous growths, and [2] not only is the seat of origin of the two forms of growth quite different, but the epithelium of the glandular growths, so far from being subservient to its connective tissue basis, is very active in pushing itself in all directions, forming acini and cysts and secreting a dense fluid, in papillomatous growths. Though vessels abound and increase of papillary projections progresses actively, none of the more complicated processes take place, and the fluid contents of the cysts in which they grow are almost devoid of organic products. These cysts are thus supposed to grow from Wolffian tube relics in the hilum. Pfannenstiel,[1] Marchand, Flaischlen, Whitridge Williams, Kossmann and others hold the view that they may arise from Pflüger's ducts, germinal epithelium, aberrant relics of Müller's ducts or Graafian follicles; but, as previously stated, it is not quite clear that a sharp distinction can be drawn between adenoma and papilloma.

Papillomatous cysts rarely reach the huge size of the multilocular ones; they are generally bilateral and grow slowly, but may cause pressure symptoms early if they involve broad ligaments. They contain thin watery fluid, as a rule, of low specific gravity, and are generally multilocular; they may have, but often have not, a pedicle, and they tend to force themselves away from the hilum into the broad ligament, or if growing in the broad ligament they may rarely invade the hilum.[4] Such cysts invading the broad ligament are among the most difficult and dangerous to remove. They are practically sessile, and may

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[1] Doran, *op. cit.* p. 59.

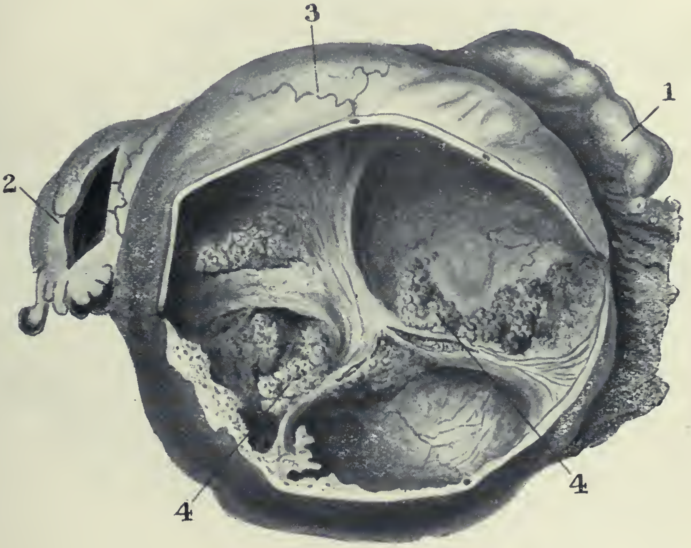
[2] Doran, *op. cit.* p. 65.

[3] Pfannenstiel, *Arch. f. Gyn.* vol. xlviii.; see also Griffith, Allbutt and Playfair, *op. cit.* p. 846, and Kossmann in Martin's *Krankheiten der Eierstöcke*, 1899, pp. 413, 929.

[4] Doran, *op. cit.* p. 68.

Fig. 94.

PAPILLOMATOUS OVARIAN CYST.



An ovarian cyst and part of the Fallopian tube. The cyst is composed of several loculi, the walls of which are studded with papillomatous growths. The Fallopian tube is dilated.

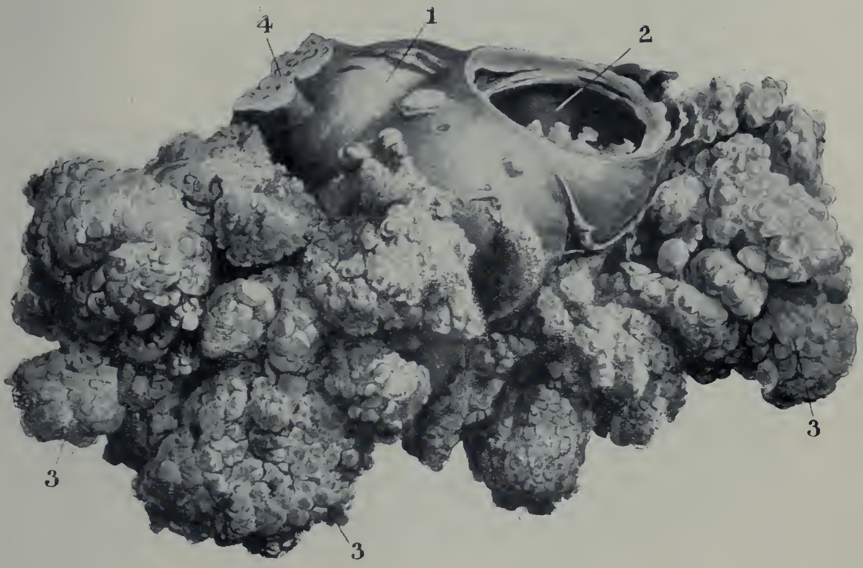
1. Uterine end of Fallopian tube. Below is the cut surface of the broad ligament.
2. Fimbriated extremity. The tube which was dilated has been opened.
3. The cyst with septa in its interior, showing its original multilocular condition.

4, 4. Papillomatous masses.

(Specimen 2913 A. Museum, St. Bartholomew's Hospital.)

Fig. 95.

PAPILLOMATOUS OVARIAN TUMOUR.



1. Remains of ovary partially converted into a cyst, which has burst.
2. Cystic portion of ovary. Its interior is filled with papillomatous growth.

3, 3, 3. Papillomatous masses in which the ovary is embedded. Many of these have sprouted through the cyst wall.

4. Pedicle.

The disease was bilateral; it occurred in a woman of 28. There were no deposits in the peritoneum.

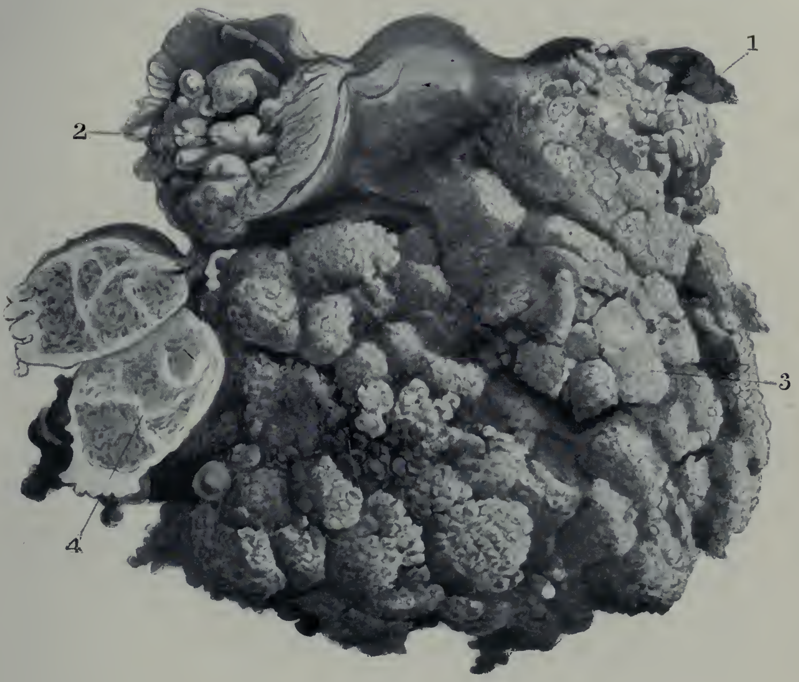
The growth consists of pure myxomatous tissue lined with columnar epithelium. There is no evidence of carcinoma.

(Specimen 2913 c. Museum, St. Bartholomew's Hospital.)

Fig. 96.

PAPILLOMATOUS DISEASE OF THE OVARY.

(Probably due to a Burst Cyst.)



A similar specimen to fig. 95. It shows portion of an ovary and Fallopian tube. The ovary is enlarged, and its whole surface is studded with numerous compound papillomatous growths. None of the normal structure of the ovary can be seen. The Fallopian tube is also enlarged, and is occupied by similar growths. The papillomatous growths have probably all arisen from the interior of an ovarian cyst which has burst, and are not true surface papillomata of the ovary.

1. Uterine end of Fallopian tube.
2. Fimbriated extremity enlarged and studded with papillomata.
3. Main mass of papillomatous disease covering the ovary.
4. Section of one of the largest masses.

The growth is a myxo-adenoma. No evidence of carcinoma can be obtained.

(Specimen 2913 B. Museum, St. Bartholomew's Hospital.)

further burrow[1] beneath the peritoneum and become irremovable. Many such cysts are malignant, as will be explained later.

The papillary growths are fragile, branching, villous processes, which easily break down (figs. 95, 96), and are very vascular. They may be fibrous in nature (figs. 92, 97), and contain the small gritty particles already spoken of, which stain deeply with hæmatoxylin (psammomata). These branching villous processes are, the writer thinks, adenomatous, and they may or may not be simple in nature. On microscopic examination all stages can be traced, from the simple branching processes, covered in an orderly manner with columnar cells which do not invade adjoining tissues, to those in which cell proliferation has gone on to an extent identical with carcinoma, and in which the cells have already invaded the stroma in all directions. There is no way of distinguishing purely adenomatous papillary growths from those which are carcinomatous except by microscopic examination after removal[2] (fig. 98).

Papillomatous cysts may contain a few papillary masses (fig. 94), or the new growth may be so luxuriant that the cyst containing it may rupture, or it may perforate the cyst-wall, and cauliflower-like masses project from the cyst into the peritoneal cavity (figs. 95, 96). Often such cysts have extremely delicate walls. The papillomatous growths may invade the neighbouring peritoneum;[3] in other cases the whole peritoneal cavity is found studded with papillomata and distended with free fluid. Doran[4] records a case of this nature in which papillomata had burrowed beneath the peritoneum till the growth had reached the common iliac arteries. In spite of this peritoneal invasion of papillomata and their seeming malignancy, after removal of such a cyst by operation, these papillomata have been known to disappear, and some cysts at first sight malignant have proved removable.[5] These burst papillomatous cysts (figs. 95, 96) are of great interest from this point of view, but all stages may be found verging towards hopeless malignancy.[6]

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[1] Martin, *op. cit.* p. 424, gives Olshausen's statistics showing how often papillary cysts burrow.

[2] Herman, *Diseases of Women*, p. 737.

[3] Bland-Sutton, *Surgical Diseases of Ovaries &c.* p. 87; and Pye-Smith, 'Hydroperitoneum with Papillomatous Cysts,' *Trans. Path. Soc.* vol. xliv. p. 111.

[4] Doran, 'Tumours of the Ovary,' *op. cit.* p. 70.

[5] Bland-Sutton, *op. cit.* p. 99; and Tait, *Diseases of Ovaries*, 2nd ed. p. 147.

[6] Länderer, quoted by Griffith, Allbutt and Playfair, *op. cit.* p. 841.

In the early stages of papillomatous cysts the ovarian tissue can be easily recognised and is only involved later, while in broad ligament papillomatous cysts, which are rare, it may be merely pushed aside. If such cysts burst, papilloma may infect the ovary or even cover it in, and cases occur where from this cause both ovaries appear to consist of dense masses of papilloma. Careful examination of such cases generally proves the condition to be one of burst cyst, and not a surface papilloma of the ovary proper, which is very rare,[1] and which is described by Whitridge Williams [2] as arising from the germ epithelium, and associated with abundant hydroperitoneum.

Malignant Cysts.—As before explained, it is extremely difficult to state before microscopic examination whether a tumour be innocent or malignant, and even the term malignancy is capable of many interpretations. More than half the cases of ovarian tumours with papillary growths [3] are adenocarcinomatous, at all events in some parts of the tumour (fig. 98), while in other parts the simple adenomatous type is retained. In malignant cysts the papillary processes become covered with many layers of atypical proliferating cells which fill the secondary cyst cavities, and further make their way into the stroma in invading columns, exactly imitating, if not identical with, a carcinomatous process. Not only does this occur, but the stroma of the tumour itself takes on a malignant type, for in rapidly growing cysts of this nature the firm fibrous arrangement gives place to tissue of an embryonic character, with rudimentary vessels, and undergoing a change which is to all intents and purposes sarcomatous; this may be another method of malignant dissemination. In such cases, clinically at least, malignancy can only be suspected by their rapid growth. In more than two-thirds of the cases they are bilateral,[4] and they displace adjoining parts and invade the retroperitoneal tissues. Many are completely sessile; they break down at the slightest touch, often with much bleeding, and are some of the most formidable and distressing varieties of ovarian tumours. They cause wasting and hydroperitoneum, and may give rise to metastatic deposits [5] in the viscera, such as the liver,

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[1] Doran, *op. cit.* p. 66; Bland-Sutton, *op. cit.* p. 97.

[2] Whitridge Williams, *Johns Hopkins Hospital Reports*, vol. iii. 1893.

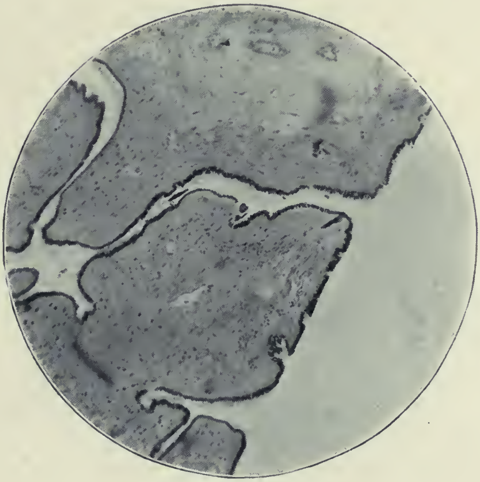
[3] Herman, *Diseases of Women*, p. 138.

[4] Herman, *op. cit.* p. 739.

[5] Herman, *ibid.*

Fig. 97.

FIBROUS PAPILLOMATA FROM THE INTERIOR OF AN OVARIAN CYST.



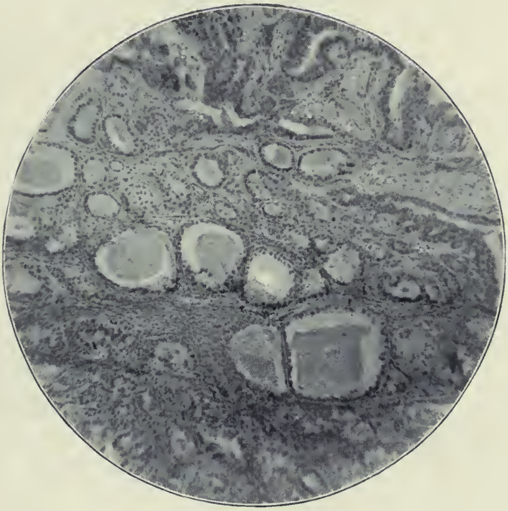
Section of a portion of an ovarian cyst, to show the hard papillomatous growths which sprung from its interior.

The masses consist almost of pure fibrous tissue projecting into the cyst cavity (to the right of the section). Each is covered with a single layer of columnar epithelium. In some places in the section this epithelium is almost absent.

(Microphotograph by C. H. Cosens.)

Fig. 98.

PORTION OF A MALIGNANT OVARIAN CYST.



Section of the solid portion of the base of an ovarian cyst which had hopelessly invaded the pelvis, and was irremovable.

The great portion of the section shows gland-like spaces filled with mucin and lined with columnar cells. In other areas the columnar cells are not confined to definite limits, but are rapidly proliferating, and invading the stroma in columns and areas identical with that seen in carcinoma.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

uterus, stomach, or omentum, as well as in periosteum of the ribs, vagina, and retroperitoneal glands.

Ovarian Dermoids.[1] (Figs. 99, 100, 101.)—These are tumours furnished with skin or mucous membrane, occurring in situations where such structures are not found under normal conditions.

Bland-Sutton[2] has devoted much time and labour to the subject, and his work on 'Dermoids' should certainly be consulted.

The amount of skin in a dermoid varies in different cysts; also the complexity of the cutaneous appendages with which it may be furnished. Sometimes the whole cyst is dermoid, in others dermoid structures may only occupy one loculus of an ordinary multilocular cyst.

Hair, sebaceous glands, sweat glands, teeth, structures like mammæ (Bland-Sutton), horn, nail, bone, unstriped muscle, and tissue identical with brain matter, may be found in dermoid tumours of the ovary. 'It is not surprising that so many tissues are found promiscuously in these cysts if they be really developed from an unimpregnated ovum (Graafian follicle), for nobody can doubt that the ovum possesses the germ of all tissues.'[3]

The hair in dermoids varies in length and amount. Occasionally only a few hairs occur (figs. 99, 100), or it may be rolled up and fill the cyst; it is sometimes very long, and mixed with the pultaceous greasy matter commonly found in these cysts, which is really fluid fat, for it solidifies like butter after its removal from the cyst. Fat may be found in little round pill-like masses,[4] each containing a hair, the pill-like shape being possibly due to some peculiar constitution of the fat[5] (fig. 101). Cholesterine crystals and epithelial débris are common; sebaceous glands occur and are generally well marked, as are also sweat glands; they generally occur in clusters.

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[1] Herman, *op. cit.* p. 739.

[2] Bland-Sutton, *Tumours, Innocent and Malignant*; *id. Surgical Diseases of Ovaries and Fallopian Tubes*, 1891, p. 57; *ibid.* 2nd ed. chaps. viii. ix.; also Delbet, 'Kystes Dermoïdes,' *Traité de Chirurgie*, 2nd ed. vol. viii. p. 355, Paris, 1899; also Wilms in Martin's *Krankheiten der Eierstöcke*, 1899, p. 576–607.

[3] Doran, *Diseases of Ovaries*, 1884, p. 88.

[4] Butler-Smythe, *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 15.

[5] Doran, *Diseases of Tube and Broad Ligament*, 1884, p. 80. See also Bland-Sutton, *Surgical Diseases of Ovaries, &c.* p. 65, in which a dermoid contained 3930 epithelial balls (Dr. W. Walter).

Bony plates, and knots or pegs of hyaline cartilage (fig. 102), may also be found. The bony plates (fig. 103) may be irregular, resembling the facial bones of teleostean fishes.[1] In some cases bone is found covered with a soft membrane like periosteum, and teeth may grow from such membrane where no bone exists (fig. 99). Heavy nodular masses of ivory-like bone have been described by Spencer Wells.[2]

Numerous cystic or tubular structures occurring in the walls of dermoid cysts are described by Doran.[3] Some lined with large columnar cells are like small glandular ovarian cysts, others simulate mucous glands.

Nerve tissue[4] is sometimes found including medullated fibres, neuroglia, and ganglion cells of varying sizes.

Plain muscular fibre is frequent, but striated never occurs. Ovarian tissue resembling **mammæ** is described by Bland-Sutton,[5] with nipple-like projections (fig. 100) beset with sebaceous glands. Some of these 'mammæ' may contain glandular tissue like that of the normal breast, but the cells are more cubical in shape; even fluid like colostrum has been described filling a cyst in such an ovarian mamma (Shattock). Probably most of the tissue in these tumours is fat, and the glands described are modified sebaceous and sweat glands.[6]

Horn and nail-like tissue have been noted. Kanthack described[7] epithelial pearls in dermoids.

Teeth[8] (fig. 99, 103) are fairly common in ovarian dermoids and may occur in large numbers; 400 have been counted in a single cyst, they may be embedded in loose bone like an alveolus, or not. They generally occur in groups, and may resemble incisors, canines, or supernumerary teeth. They may have single or multiple cusps and have short roots, which are generally single. The enamel and dentine are often well formed; the pulp is irregular and imperfect. The presence of nerve tissue in the pulp is said sometimes to occur.

An interesting point with regard to dermoid tumours is the question of their *malignancy*. Doran[9] says this occasionally

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[1] Doran, *op. cit.* p. 85. [2] *Ibid.* p. 87. [3] *Ibid.* p. 88.

[4] Bland-Sutton, *op. cit.* p. 66.

[5] Bland-Sutton, *op. cit.* 1891, p. 58.

[6] Griffith, Allbutt and Playfair, *op. cit.* p. 840.

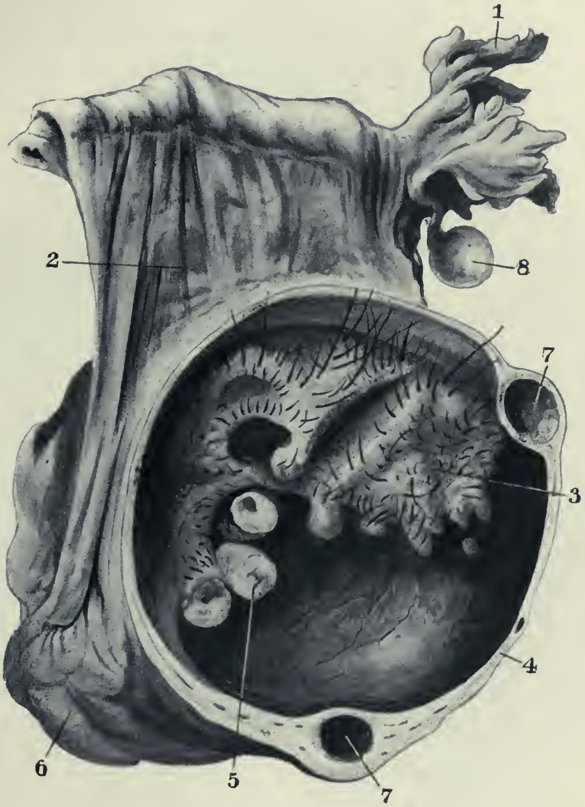
[7] Kanthack, *Journ. Anat. and Physiol.* vol. xxv.

[8] Bland-Sutton, *op. cit.* p. 63; *id. Trans. Odontolog. Soc.* vol. xxii. p. 156.

[9] Doran, *op. cit.* p. 90.

FIG. 99.

A DERMOID CYST OF THE OVARY CONTAINING TEETH.



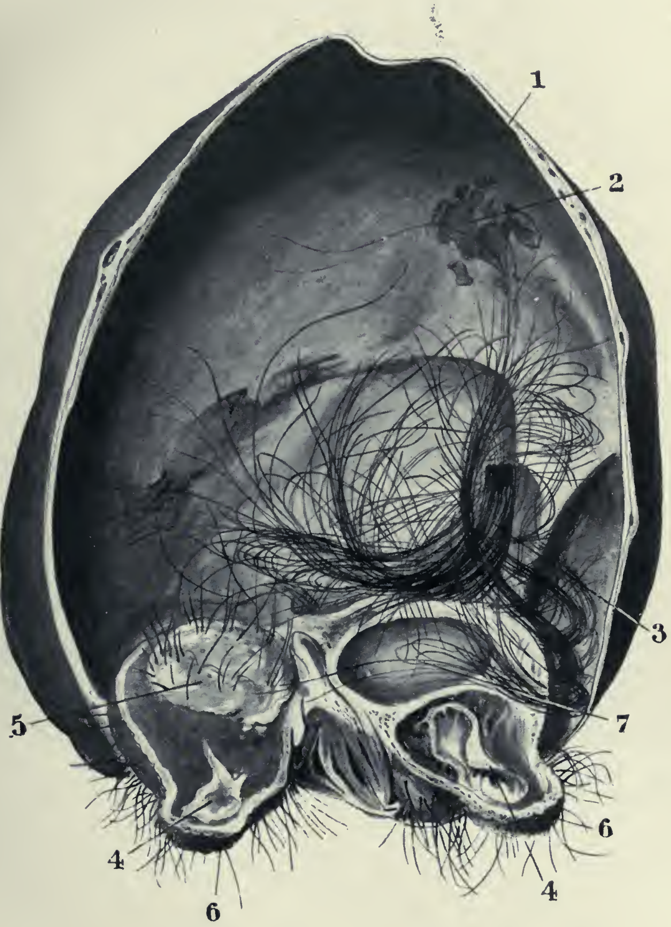
An ovarian cyst with thick walls. The interior is occupied by a raised area of soft tissue covered with true skin, numerous hairs project from the surface, and in places nipple-like processes are seen. Below and to the left are three well-marked teeth.

1. Fimbriated extremity of the tube.
2. Meso-salpinx.
3. Mass of skin, hairs, and nipple-like projections.
4. Cyst wall.
5. Three well-formed teeth.
6. Remains of ovarian tissue.
7, 7. Secondary cysts in the wall of main cyst.
8. Pedunculated cyst of the fimbriæ (hydatid of Morgagni).

(Specimen 2373. Museum, St. Thomas's Hospital.)

FIG. 100.

DERMOID CYST OF THE OVARY (CONTAINING HAIR, BONE, AND A NIPPLE-LIKE PROCESS).



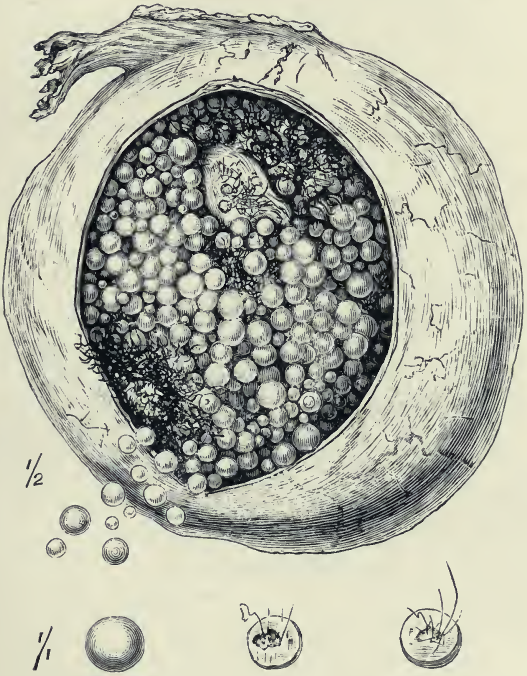
Portion of a large dermoid cyst of the ovary. It contains hair and a curious nipple-like process. This is covered with skin, but nothing like ducts or breast tissue was found on microscopical examination. It consists chiefly of fat.

1. Main cyst wall.
2. Plates of calcareous material.
3. Long masses of hair.
4, 4. Bony plate contained in the nipple-like process which has been cut in half.
5. Pad of fatty material fitting into a hollow (7) on the opposite side.
6, 6. Extremity of the so-called nipple, covered with true skin and hair.

(Specimen 2373 A. Museum, St. Thomas's Hospital.)

Fig. 101.

DERMOID TUMOUR OF THE OVARY.

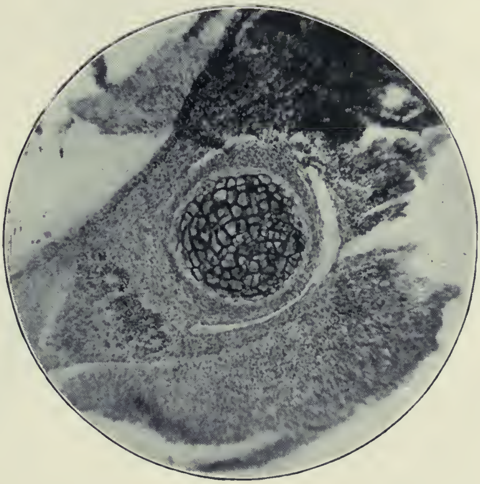


An ovarian dermoid containing pill-like masses of epithelial balls, each of which contains one or more hairs as a nucleus. Over 3,000 balls were counted in the above specimen.

(Bland-Sutton, ‘Surgical Diseases of the Fallopian Tubes,’ new edition, p. 65.
W. Walter’s case.)

Fig. 102.

HYALINE CARTILAGE IN AN OVARIAN DERMOID.

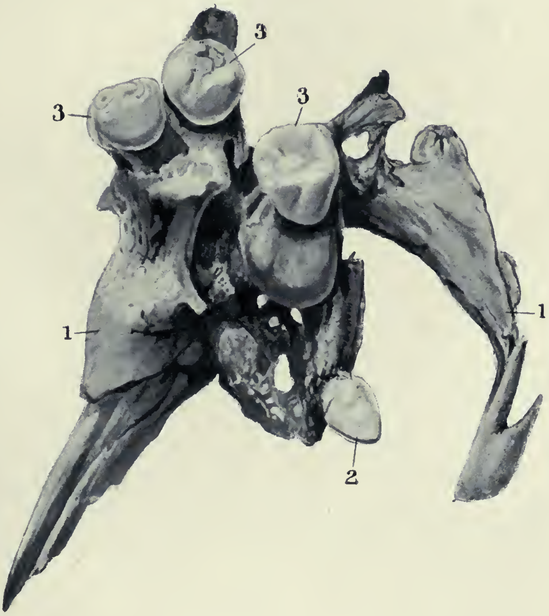


Section of a portion of the wall of a dermoid cyst of the ovary. In the centre is seen a rounded area of hyaline cartilage.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 103.

TEETH FROM AN OVARIAN DERMOID.



An irregular branching piece of bone contained in a dermoid cyst of the ovary, in which are implanted well-formed teeth.

1, 1. Bony mass.
2. A tooth resembling a canine of the first dentition.
3, 3, 3. Teeth resembling molars.

(Specimen 2376. Museum, St. Thomas's Hospital.)

occurs in the form of sarcomatous growth, which develops from the abundant connective tissue of such cysts; but it is very difficult to venture a decision on any quasi-cancerous tissue in cysts of this kind.[1]

Dermoids[2] may very rarely be disseminated in the peritoneal cavity after rupture. They may occur at any period of life, and have been recorded soon after birth; Bland-Sutton doubts if they can occur at birth. They have been found in very old women. As a rule they grow slowly and do not often attain a large size. They are very liable to suppurate and are dangerous cysts. If they burst into viscera very troublesome sinuses result.

They are extremely interesting in connection with pregnancy,[3] with which they not infrequently occur. Owing to their slow growth, the period through which a patient with a dermoid incurs the risk of pregnancy is longer than is the case with a patient who has a rapidly growing tumour. Pregnancy may occur where both ovaries are converted into dermoid cysts. Generally dermoid tumours are unilocular, but they may occasionally contain several cavities, or they may occur among the loculi of an ordinary multilocular cyst. About 3·5 per cent. of all ovarian tumours are dermoid[4]

Solid Tumours of the Ovary.[5]—These are not common and form only about 5 per cent. of all ovarian tumours. They may be innocent or malignant, and may undergo cystic degeneration. They are often bilateral, and are frequently associated with hydroperitoneum.

Fibromata.[6] (Figs. 104, 105.)—These are certainly very rare. Doran has described tumours of the ovary practically identical with fibroids of the uterus. They may occur either in the ovary or the ovarian ligament; in the ovary they form hard painless tumours, and may occur in quite young women. Many tumours diagnosed as 'fibroids' in young women are probably fibroids of the ovary. Fibroids of the ovary are liable to calcify[1] (fig. 104), and torsion of the pedicle may occur. Free fluid in the peritoneum was noted in five out of eleven cases by Doran.[2] On section ovarian fibromata consist of characteristic wavy bundles of fibrous tissue packed together, as in ordinary uterine fibroids.

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[1] Bland-Sutton, *Surgical Diseases of Ovaries &c.* p. 77, denies that there is any proof at present of the malignancy of dermoids. See also H. Kelly, *Operative Gynæcology*, vol. ii. p. 280, and Wilms, *loc. cit.* p. 606.

[2] Bland-Sutton, *op. cit.* p. 70; Griffith, *op. cit.*, who quotes Martini's case, in which the peritoneum was studded with secondary dermoids, p. 841.

[3] Herbert Spencer, *Trans. Obstet. Soc. Lond.* vol. xl. 1898, pp. 14, 259; McKerron, *ibid.* vol. xxxix. 1897, p. 334.

[4] Griffith, *op. cit.* p. 840.

[5] Olshausen, quoted by Griffith, Allbutt and Playfair, *op. cit.* p. 842. Herman, *Diseases of Women*, p. 745, says 'only 1 to 2 per cent. of all ovarian tumours are solid.'

[6] Doran, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 39; *ibid.* vol. xxxviii. 1896, p. 187; *ibid.* vol. xxx. 1888, p. 419.

Fibroma of the Ovarian Ligament.—Doran[3] records three cases. It is a very rare disease. In two cases the disease was fibroma with cystic cavities; the third was doubtfully sarcomatous, and weighed over sixteen pounds. Œdema and softening may occur; ascites and adhesions are rare, and menstruation is not affected.

Myomata of the Ovary.—These are described by Bland-Sutton as composed mainly of unstriped muscle fibre, or a mixture of muscle and fibrous tissue—fibro-myoma. The question arises whether myoma and fibroma are not practically the same tumours; however, fibro-myoma seems certainly to be one of the commonest forms of solid innocent ovarian tumours. They may grow to a large size.

Sometimes, in an ovary, several hard fibrous nodules may be found; they are usually discovered accidentally or in the post-mortem room. Generally they are small oval enlargements as big as a bean, and are probably corpora lutea, which have undergone hypertrophy instead of atrophy.[4]

Similar fibroid changes frequently occur in ovaries, generally arranged concentrically round old follicles. On microscopical examination, the fibrous tissue presents a wavy appearance, and stains very deeply, while in the neighbourhood the vessels appear to be in a condition of arteritis obliterans. The writer believes these are the tumours called *gyromata* described by Mary Dixon Jones[5], and that the condition is due to degenerative changes in the fibrous tissue around old contracting corpora lutea.

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[1] Roberts, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 8.

[2] Doran, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 188; also Cullingworth *ibid.* vol. xxxix. 1897, p. 280.

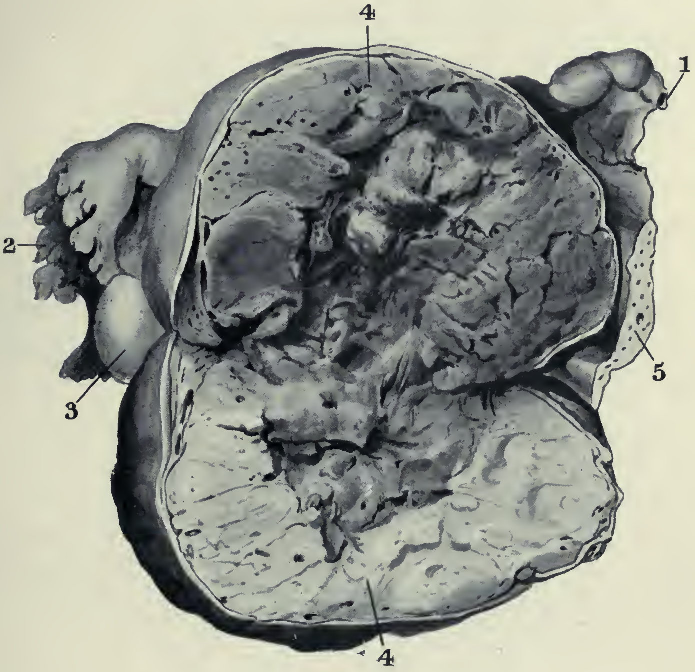
[3] Doran, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 189.

[4] Griffith, Allbutt and Playfair, *op. cit.* p. 843.

[5] M. Dixon Jones, *New York Med. Journ.* May 1890.

Fig. 104.

CALCIFYING FIBROMA OF THE OVARY.



A tumour growing from the ovary, which is irregularly ovoid in shape. Its cut surface shows the tumour to be a calcifying fibroma; irregular calcareous plates and spicules are scattered throughout its substance. In the centre the tumour is breaking down and œdematous. No dermoid structures are present. There was no hydro-peritoneum. Microscopically the tumour is a fibroma; it weighed 6¼ ounces, and was removed from a woman of 29.

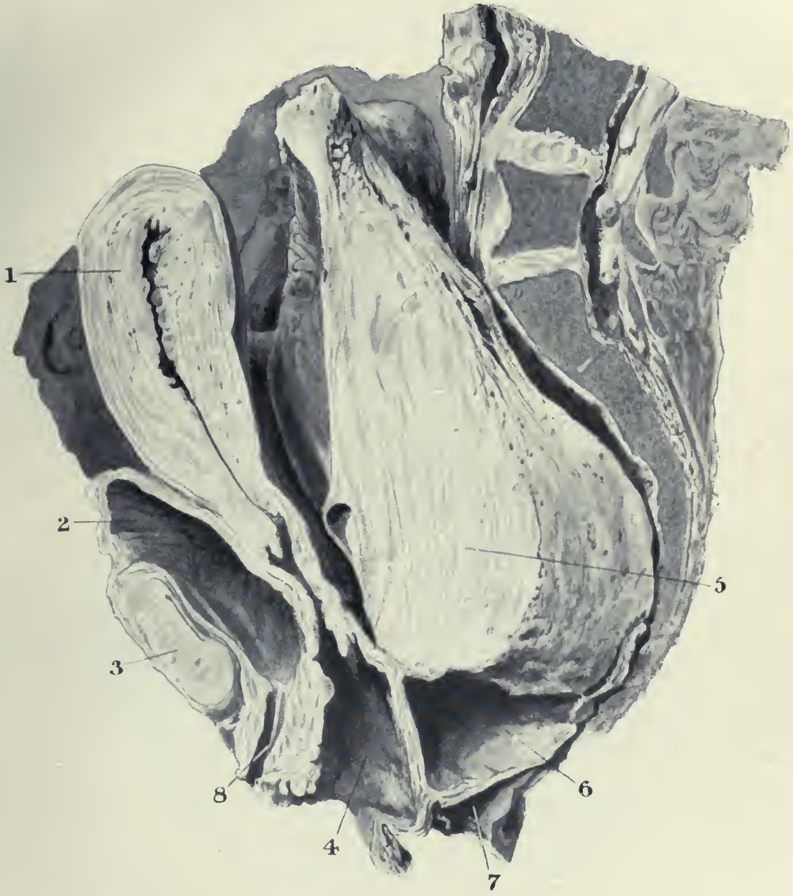
1. Uterine end of Fallopian tube.
2. Fimbriated extremity.
3. Small cyst of the broad ligament.

4, 4. Cut surface of tumour, to show central areas of degeneration.
5. Pedicle.

(Specimen 2952 c. Museum, St. Bartholomew's Hospital. Roberts, 'Trans. Obstet. Soc. Lond.' vol. xxxix. 1897.)

Fig. 105.

FIBROMA OF THE OVARY WHICH OBSTRUCTED LABOUR.



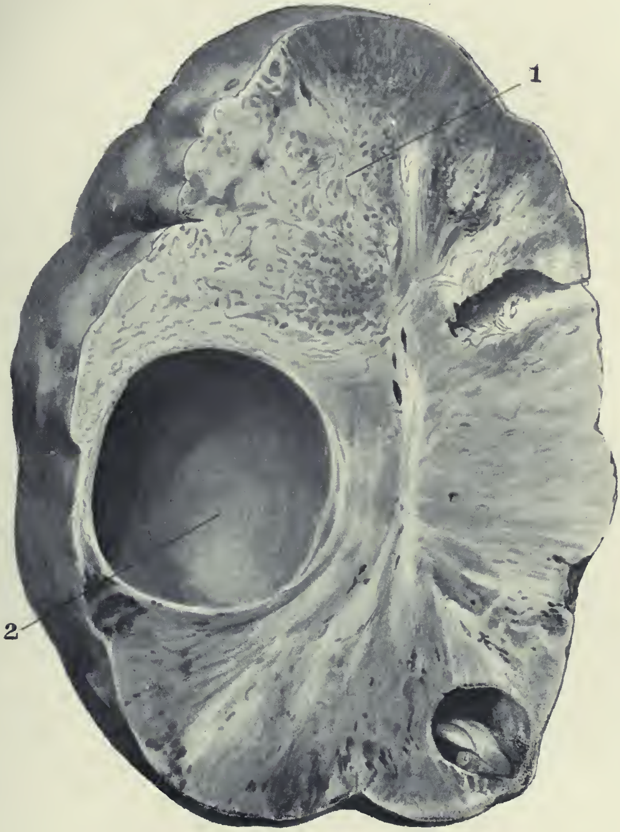
Section of a pelvis the cavity of which is occupied by a large solid tumour of the right ovary, and which had displaced the uterus upwards. It occupies the utero-sacral pouch; and during delivery was taken to be the head of an extra-uterine fœtus. The woman was delivered by version after craniotomy.

1. Uterus recently delivered, showing placental site.
2. Bladder.
3. Symphysis pubis.
4. Vagina.
5. The tumour (fibroma of the right ovary; it measured 9 in. × 4 in. × 3½ in.).
6. Douglas's pouch.
7. Rectum.
8. Urethra.

(Specimen 3091 A. Museum, St. Bartholomew's Hospital. Griffith, 'Trans. Obstet. Soc. Lond.' vol. xxxiii. 1891, p. 140.)

Fig. 106.

PRIMARY SARCOMA OF THE OVARY.



One half of a large sarcomatous mass into which the whole ovary has been converted. It is very soft, vascular, and semi-gelatinous on section. There were secondary growths in the stomach. The growth is a myxo-sarcoma.

1. Main mass of the tumour, to show the soft gelatinous material into which the ovary has been converted.
2. A large cystic space which was filled with thick fluid; a smaller one exists at the right lower border of the tumour.

(Specimen 2927 B. Museum, St. Bartholomew's Hospital.)

Leopold gave the name **endothelioma**[1] to a tumour of the ovary which resembled a fibroma, but contained alveolar spaces packed in the epithelioid cells. He traced the origin of the spaces to dilatation of lymphatic and capillary channels, with proliferation of their endothelium. This is also stated to be the origin of some forms of sarcoma of the ovary.[2]

Sarcoma of the Ovary.[3] (Fig. 106.)—This tumour is certainly rare, and as a rule both ovaries are affected. Sarcoma occurs in the ovary in the round or spindle-celled form. It is a very malignant form of disease, and grows rapidly. Cases of supposed sarcoma must be investigated with great care; many specimens described as sarcomata may be fibromata. Secondary sarcomatous growth in the lungs, liver, stomach, and peritoneum would probably settle the question of a doubtful tumour.

The malignancy of some papillomatous cysts and dermoids may be due to sarcomatous changes in their stroma, but the whole question of such 'malignancy' is at present unsettled.

Sarcomata occur in fœtuses and children,[4] but they differ somewhat from the tumours of adults, in repeating the embryonic characters of the connective tissue of the fœtal ovary, and cell elements preponderate. Bland-Sutton proposes the name 'Oöphoroma' for these growths. Doran[5] has described a tumour of this class in a seventh months' fœtus.

Congenital Ovarian Cysts may be unilocular or multilocular, unilateral or bilateral; there are several such tumours in the Royal College of Surgeons' Museum (see fig. 83). Solid tumours occur in greater frequency in fœtuses than in adults, and not a few are malignant.

Carcinoma of the Ovary.[6] (Figs. 107, 108.)—The majority of cases of cancer of the ovary are probably secondary, and occur with cancer of the breast (fig. 108) or uterus. Occasionally, melanotic deposits are seen; there is one such specimen in St. Bartholomew's Hospital Museum, but this is probably a sarcoma. Secondary deposits of cancer are not common, but both ovaries may be affected; in such cases large, soft, solid tumours are found. Bland-Sutton,[1] recording the Middlesex Hospital post-mortems, found that out of 89 cancer cases the ovaries were affected five times. The author has sections of a case where 'oöphorectomy' was performed for cancer of the breast, and at the operation the ovaries themselves were found to be the seat of secondary deposits of scirrhous carcinoma (fig. 108).

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[1] Griffith, *op. cit.* p. 843; and V. Velitz, *Zeit. für Geb. und Gyn.* Bd. viii.

[2] Amann, *Arch. für Gyn.* 1894, p. 484.

[3] Cullingworth, *Trans. Obstet. Soc. Lond.* vol. xxi. 1879, p. 276, and drawing.

[4] Bland-Sutton, *Surgical Diseases of Ovaries &c.* new ed. 1896, p. 43; *op. cit.* 1st ed. p. 87, 'Table of Cases of Ovarian Tumours in Infants and Girls under Fifteen.'

[5] Doran, *Trans. Path. Soc.* vol. xl. p. 300.

[6] Doran, *Diseases of Ovaries*, 1884, p. 103.

There is no doubt that primary carcinoma[2] (fig. 107) of the ovary occurs. Griffith[3] exhibited a well-marked case at the Obstetrical Society in February 1900. The ovary was transformed into a large solid mass, which had rapidly developed. The patient was aged 58. Sections exhibited well-marked columnar carcinoma; the tumour was pedunculated and was free from adhesions; the other ovary was not affected.

Shattock, quoted by Bland-Sutton,[4] reports a similar case of a large lobulated tumour measuring eleven inches by five, consisting on section of tortuous loculated spaces lined with tall, slender, closely compressed columnar epithelium. Much interest attaches to the relation of such tumours to the adenomatous growths of the ovary. It is reasonable to believe that, where adenoma occurs, carcinoma may also be present. Certainly the examination of some of the solid portions of so-called multilocular glandular or papillary cysts has led the author to conclude that malignant characters, at all events under the microscope, occur much oftener than is supposed. Some of the sessile broad ligament cysts, which are bilateral and irremovable, are undoubtedly malignant, and even in simpler adenomatous cysts one portion may appear innocent while sections taken from another portion of the *same* tumour cannot be differentiated from cancer. The life history of such growths is worthy of the closest study. For long periods they seem to confine their proliferations within their own cyst-walls, but the invasion of adhesions or of the pedicle may occur, and such a process is cancerous. Secondary deposits in the omentum and peritoneum seem to be more commonly due to the disease spreading through adhesions than through the pedicle.

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[1] Bland-Sutton, *Surgical Diseases of Ovaries &c.* 1891, p. 81.

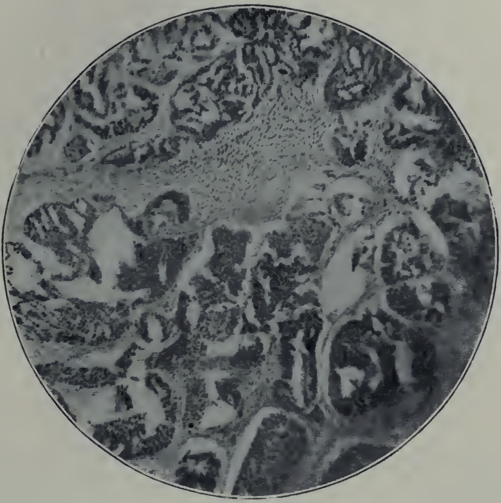
[2] Martin, *loc. cit.* p. 541. He reports 74 per cent. of his cases of primary malignant ovarian tumour as 'carcinoma' (valuable tables given).

[3] Griffith, *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 34.

[4] Bland-Sutton, *op. cit.* p. 77; Shattock, *Trans. Path. Soc.* vol. xl. p. 208.

Fig. 107.

PRIMARY CARCINOMA OF THE OVARY.

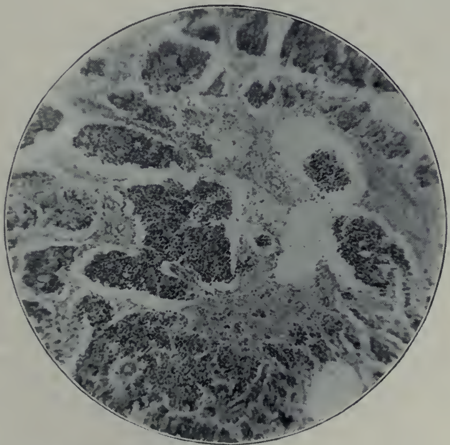


Section of a cancerous tumour of the ovary of a primary nature. The ovary is transformed into a mass of cancer; irregular spaces are seen filled with atypical columnar epithelium undergoing rapid proliferation. Many of the cells in the upper part of the section are invading the stroma in irregular columns.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

Fig. 108.

CARCINOMA OF THE OVARY, SECONDARY TO SCIRRHUS OF THE BREAST.

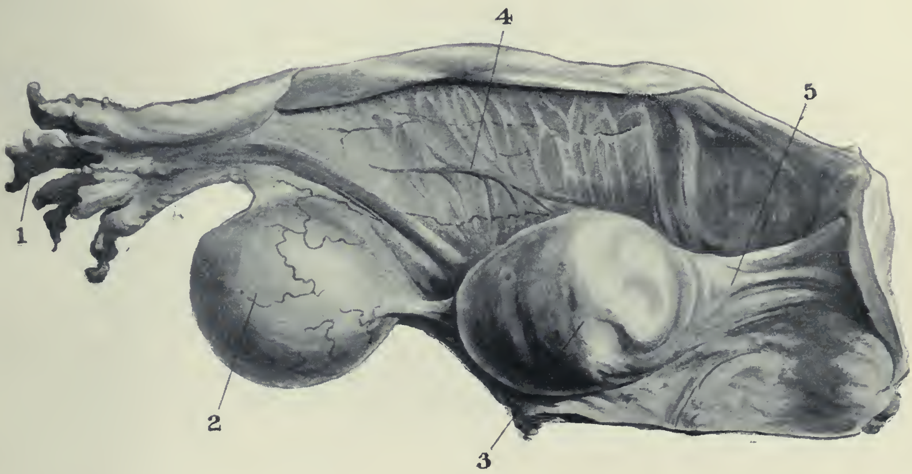


Section of an ovary which has been converted into a mass of carcinoma. The disease was secondary to cancer of the breast. The ovary is everywhere occupied by masses and groups of carcinomatous cells, more or less contained in irregular acini. The operation was undertaken in the hope of influencing a mass of recurrent growth after removal of the breast for cancer, but both ovaries themselves were found involved.

(Microphotograph by C. H. Cosens.)

Fig. 109.

SMALL CYST OF THE BROAD LIGAMENT (DISTINCT FROM THE OVARY).

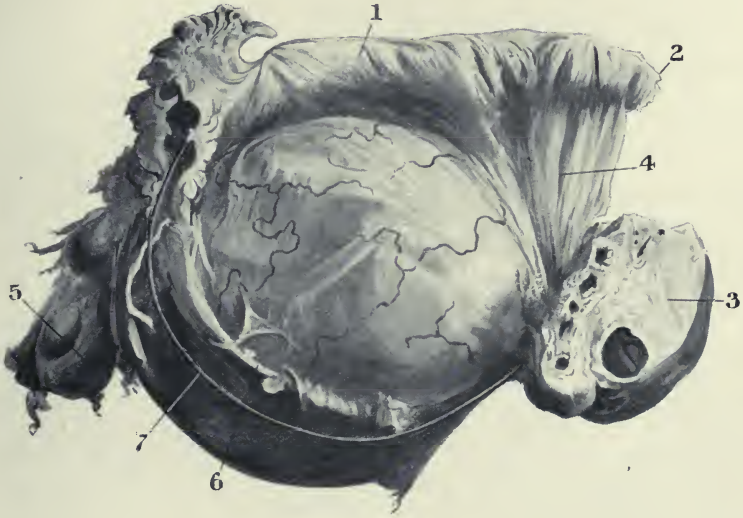


1. Fimbriated extremity of the Fallopian tube.
2. Small simple cyst developed in the outer border of the broad ligament, the layers of which cover the cyst.
3. Ovary (normal).
4. Remnants of the parovarium.
5. Ligament of the ovary.

(Specimen 2403. Museum, St. Thomas's Hospital.)

Fig. 110.

CYST OF THE BROAD LIGAMENT (PAROVARIAN).



Fallopian tube, part of the ovary, and intervening portion of the broad ligament. The ovary is small and apparently healthy. A thin-walled cyst, 2 inches in diameter, lies between the folds of the broad ligament; the ovarian fimbria is stretched over it. Two small cysts hang from the fimbriated extremity of the tube.

1. Fallopian tube. Note the folds of the broad ligament passing from it to form the capsule of the cyst.
2. Uterine end of tube.
3. Ovary (normal).
4. Folds of broad ligament.
5. Two small accessory cysts of the fimbriæ.
6. Ovarian fimbria passing from the tube to the ovary. A bristle (7) is passed along it.

(Specimen 4587 b. Museum, Royal College of Surgeons.)

Shattock, quoted by Griffith,[1] reports a case in which the cancerous process arose in the lining of the Graafian follicle.

More than half the cases of primary cancer are bilateral.[2] The medullary glandular type is probably the commonest. It seems to occur either in the young below twenty, or in very old women.[3]

Broad Ligament Cysts. (Figs. 109, 110).—A variety of cysts may be found occupying the broad ligament. Tumours[4] may arise primarily in this situation, either true parovarian cysts, or cysts of the broad ligament developed outside that structure, possibly from Gartner's duct low down beside the uterus. Rarely, cysts which arise in the ovary, near the hilum, may invade the broad ligament; thus papillary and dermoid tumours may be found in this situation. It appears that broad ligament cysts rarely invade the hilum itself.[5]

The cysts which arise in the broad ligament are distinguished from ordinary ovarian cysts by usually having no pedicle, unless very large, and by being encapsuled[6] and covered in by the layers of the broad ligament itself (fig. 110), sometimes much thickened, which can be seen to be quite distinct, and to move over the thin-walled cyst below. Sometimes the muscular fibres of the peritoneum can be seen running over such cysts. It is aptly termed by Champneys the 'Uterine Platysma.' This layer has to be divided before such cysts (which are not sufficiently pedunculated to be tied off) can be enucleated, an important point to remember at operations. After removal of such a cyst these muscular layers of the broad ligament retract and considerably diminish the size of the cavity left. Many such cysts are completely free from the ovary (figs. 109, 110), or the ovary may be found stretched over or flattened against the cyst-wall. The Fallopian tube passing over the top of such cysts is sometimes enormously lengthened. Generally broad ligament cysts are unilocular, and contain clear watery fluid of low specific gravity and no albumin. They may burrow very widely in the broad ligament dipping down at the side of the uterus, and these thin deep pockets render their enucleation sometimes very troublesome.

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[1] Griffith, Allbutt and Playfair, *op. cit.* p. 844.

[2] Martin, *loc. cit.* p. 541 (valuable statistics given).

Herman, *Diseases of Women*, p. 750.

Coblenz, *Virchow's Arch.* vol. lxxxiv. p. 26.

[5] Doran, *Diseases of Ovaries and Tubes*, 1884, p. 68.

[6] Doran, 'True and False Capsules in Ovariotomy,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 265.

Much discussion has arisen, with regard to broad ligament cysts,[1] as to whether they all arise from the parovarium proper, or are developed outside it, so that the name 'parovarian' cannot be accurately applied to them. Hence it is better to use the term 'simple cyst of the broad ligament' than to employ the term 'parovarian cyst' when speaking of a large cyst free from the ovary. Such cysts up to the present time have been considered to be invariably parovarian.[2]

These simple broad ligament cysts (the so-called parovarian) may sometimes grow to a large size, containing even pints of fluid. They are not seen before the age of sixteen, due according to Bland-Sutton[3] to the 'stimulation of the parovarium' by the advent of puberty, though Doran[4] says the parovarian tubes and everything associated with them tend to atrophy at this period of life, and not to enlarge. If pedunculated they may undergo axial rotation. They are generally harmless tumours, owing to the simple fluid they contain.

Small pedunculated cysts of Kobelt's tubes, and small subserous collections of fluid so frequently seen beneath the broad ligament, are unimportant, as they rarely become large.[5]

Sometimes papillary cysts occur in the broad ligament, histologically (fig. 111) and pathologically identical with those growing in the hilum of the ovary. They grow rapidly between the layers of the broad ligament, the ovary being free. Doran[6] believes these cysts are derived from the vertical tubes of the parovarium and their prolongation into the tissue of the ovarian hilum. Such cysts, if once they burst, sprout freely into the peritoneal cavity, and soon grow over the tube, fundus uteri, and peritoneum; they are rare.

These papillary cysts entirely free from the ovary must not be confounded with glandular oöphoritic cysts. Doran[7] says that no case of multilocular cystic disease of the broad ligament with 'glandular intracystic growths and no implication of

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[1] Bantock, *Trans. Obstet. Soc. Lond.* vol. xv. 1873, p. 105.

[2] Doran, *op. cit.* p. 49; but see Martin and Kossmann, *loc. cit.* pp. 924 to 937.

[3] Bland-Sutton, *op. cit.* p. 107, new ed. 1896; 'Parovarian Cysts,' p. 96.

[4] Doran, *op. cit.* p. 51.

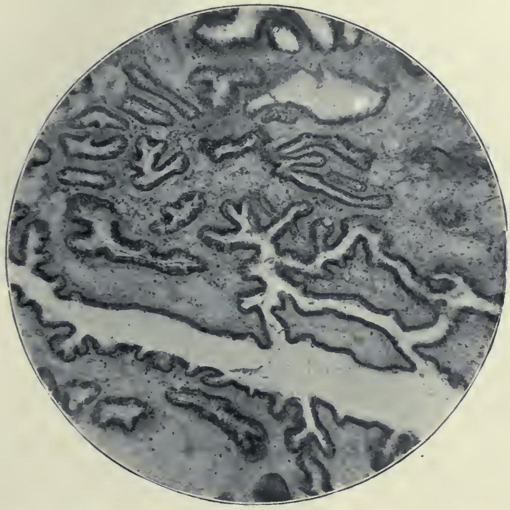
[5] The Hydatid of Morgagni is described in the chapter on 'Diseases of the Fallopian Tubes.'

[6] Doran, *op. cit.* p. 51. Kossmann, *loc. cit.* p. 935, traces them to vestiges of Müller's duct.

[7] Doran, *op. cit.* p. 50.

FIG. 111.

PAPILLOMATOUS CYST OF THE BROAD LIGAMENT.



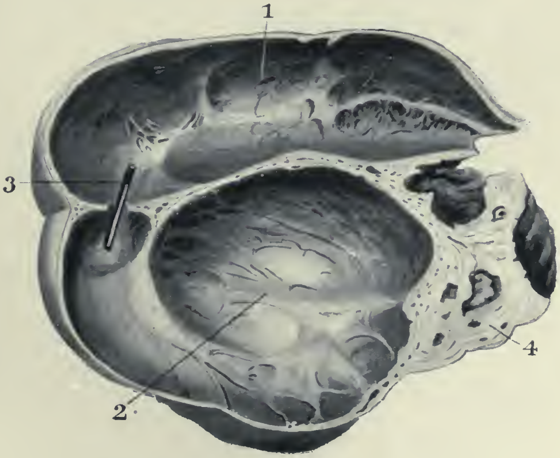
Section from the solid portion of a cyst which occupied the broad ligament. It contained many papillomatous growths in its interior.

The section shows the microscopical appearances of the intra-cystic growths. The stroma is everywhere occupied by irregular complex spaces lined by columnar epithelium (like an adenoma). In other places portions of the stroma project into the lumen of the cyst in branching papillomatous columns, but still covered by well-marked columnar epithelium.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

FIG. 112.

A TUBO-OVARIAN CYST.



One half of a dilated Fallopian tube and a small unilocular cyst of the ovary. The extremity of the tube is adherent to the ovarian cyst, and communicates with it by means of a minute orifice through which a glass rod (3) has been passed.

1. Much-dilated Fallopian tube.
2. Unilocular cyst of the ovary.
3. Small orifice of communication between the tube and the ovary.
4. Remains of normal ovarian tissue.

(Specimen 2937 c. Museum, St. Bartholomew's Hospital.)

the ovary' has ever been described, but that papillary cysts of the hilum of the ovary and those of the broad ligament are identical in origin and developed from parovarian relics.

Tubo-ovarian Cysts.[1] (Figs. 112, 113.)—These arise from fusion of obstructed tubes and ovaries subject to cystic degeneration, which is frequent in chronic inflammation of the appendages. In these cases the ovary is replaced by a cyst, generally unilocular which communicates with a distended tube. The orifice of communication, which is often large, does not necessarily represent the ostium of the tube, but may be an adventitious opening. The tube is curiously distorted and lengthened and resembles a 'retort with a convoluted delivery tube.' Probably the formation of the ovarian cyst and the dilatation of the tube are secondary and not primary factors, the permanent adhesion of the tube to the ovary being effected by adhesive inflammation, and the communication between them being generally either a primary occurrence or taking place at a very early period in the formation of the cyst.[2]

Probably very many of the so-called tubo-ovarian cysts are not ovarian at all, but really tubal, and care must be taken in investigating such specimens.

Doran[3] classes tubo-ovarian cysts as :

a. Pseudo-tubo-ovarian cysts.
b. Inflammatory tubo-ovarian cysts.
c. Teratological ovarian hydroceles.

In pseudo-tubo-ovarian cysts the tube seems to open into a large cavity beneath it at a very acute angle, and the fimbriæ appear to expand round the orifice of the tube and float in the cavity of the cyst, which may even have ovarian tissue expanded and flattened out between the cyst and uterus. The cyst is really the expanded, curiously bent and sacculated Fallopian tube (Hydrosalpinx), the largest end of which is bent beneath the less dilated uterine part of the tube. On section its plicæ are found to be effaced by pressure of the contained fluid, while in the less distended uterine portion the plicæ are well marked and project around the orifice communicating with the dilated portion, assuming the appearance of fimbriæ.[4] Hence one would conclude that the tube ended here, and that the smooth cavity beyond was a cyst of the ovary; sometimes, however, plicæ can still be traced over its interior, proving the whole to be tube.

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[1] Griffith, *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 273.

[2] Doran, *op. cit.* p. 168.

[3] Doran, article 'Fallopian Tubes,' *Encyclopædia Medica*, vol. iii. p. 435.

[4] Doran, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 3.

Inflammatory tubo-ovarian cysts are fairly common in chronic pelvic inflammation as both cystic degeneration of the ovary and dilated inflamed tubes occur, either in Hydro- or Pyosalpinx, and it is easily understood how either might communicate with the other (fig. 112), just as in severe pyosalpinx the suppurating tube sometimes communicates with an abscess in the ovary, though it is impossible to say which is the first affected. Doran[1] thinks the infection may be simultaneous.

Cases of a dilated tube communicating with a true ovarian cystic tumour are rare, but if this occur the contents of the cyst may be evacuated through the tube into the uterus (Hydrops Ovarii profluens). Much in the same way a papillomatous or cancerous tube may sometimes discharge watery fluid[2] (Hydrops Tubæ profluens). Pyosalpinx also may discharge its contents into the uterus.[3]

Bland-Sutton[4] points out the possible occurrence of ovarian hydroceles in the human female, arising in a tunic of peritoneum which occasionally invests the ovary, resembling the condition seen in hyænas, rats, and mice. Robinson's paper may also be referred to on the subject.[5]

In ovarian hydrocele the tube opens into a cyst on the posterior aspect of the broad ligament by its fimbriated extremity, which can still be recognised by ridges, spreading over the cyst-wall internally, the ovary forming part of the cyst wall or projecting into the cyst. It may discharge its contents periodically into the uterus. Ovarian hydroceles rarely contain papillomata (fig. 113).[6]

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[1] Doran, *op. cit. Encyclopædia Medica*, vol. iii. p. 436.

[2] Herman, *Diseases of Women*, p. 725.

[3] Doran, *op. cit. Encyclopædia Medica*, vol. iii. p. 436.

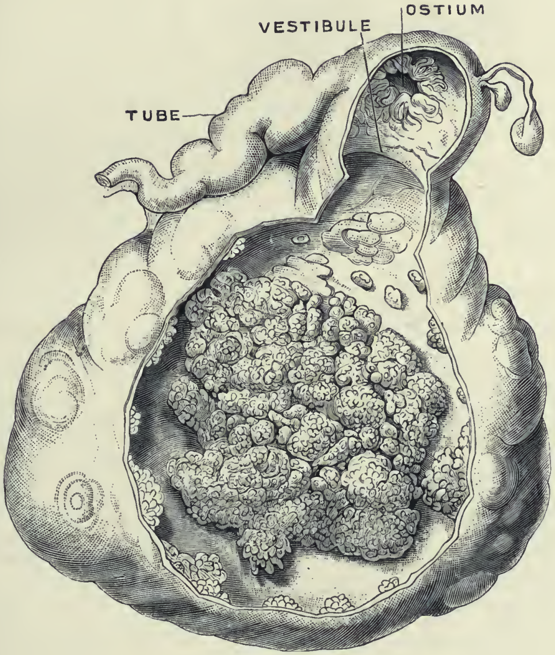
[4] Bland-Sutton, *op. cit.*, new ed. 1896, p. 102.

[5] Robinson, 'Peritoneal Relations of the Mammalian Ovary,' *Journ. Anat. and Physiol.* vol. xxi.

[6] Bland-Sutton, *Trans. Obstet. Soc. Lond.* vol. xxxiv. 1892, p. 216.

Fig. 113.

AN OVARIAN HYDROCELE CONTAINING PAPILLOMATA.



A specimen of ovarian hydrocele (Bland-Sutton) as large as a cocoanut. The Fallopian tube lies above the cyst; the uterine end is normal, but towards the cyst it becomes dilated, finally opening into it by a large circular aperture, from which folds of the mucous lining of the tube radiate, and are finally lost on the walls of a tubular chamber above the larger cyst. On the inner walls of this cyst are large masses of papillomata, identical with those found in typical paroöphoritic cysts. No trace of ovarian tissue was found.

(Bland-Sutton, 'Trans. Obstet. Soc. Lond.' vol. xxxiv. 1892, p. 216.)

SECONDARY CHANGES IN OVARIAN TUMOURS

Inflammation of the surface, the walls, or the contents may occur. The surface of a cyst which is inflamed loses its white glistening look and becomes roughened by local peritonitis. Omentum and intestines may be seen sticking to the cyst, attached by vascular adhesions, and the cyst may be of a yellowish or brownish colour with patchy ecchymoses, indicative of degenerative processes, its walls being so rotten that the slightest touch may rupture the cyst.

Adhesions may occur of all varieties. If recent, they can easily be separated or sponged away, but when old they become dense, firm bands uniting intestine, omentum, and peritoneum to the cyst, and in some cases the cyst is so hopelessly bound down that it is almost impossible to remove it.

They are the result of peritonitis, which causes adjacent surfaces to be bound together by lymph exudation, the adhesions subsequently becoming fibrous. If this fibrous organisation occurs rapidly the parts are firmly matted together, but if movement occur between parts the recent adhesions may stretch into long narrow bands and strings. Old adhesions, by becoming vascularised, may to some extent nourish the cyst wall. Parietal adhesions of the peritoneum to the cyst-wall are sometimes very puzzling, as it is difficult, in cutting down on to such a cyst, to be sure of the layers divided, and the cyst or intestines may easily be opened. Peritoneal adhesions in the pelvis, especially on the posterior aspect of a cyst, are very dangerous, for if care be not taken in separating them the pelvic blood-vessels may be opened or the viscera torn.[1]

The cyst itself and its contents may inflame ; this is not uncommon with dermoids, and with tumours which get jammed in the pelvis. Large abdominal cysts are not so likely to be affected.

The method of infection of ovarian cysts is either through the intestinal canal, the bladder, the Fallopian tube, or from tapping. The Fallopian tube in certain cases, just as in pelvic peritonitis, may be a source by means of which the cyst may become infected; and probably infection from the uterus, in cases after parturition complicated by ovarian cyst, occurs in this way.

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[1] Doran, 'Capsules, False and Real, in Ovariotomy,' *Brit. Med. Journ.* 1896, vol. i. p. 960.

The intestinal tract is another common source of infection. Pieces of intestine may adhere to the cyst wall, diffusion of intestinal gases takes place, and suppuration then occurs. Bland-Sutton[1] points out the occurrence sometimes of adhesion of the vermiform appendix to an ovarian cyst; disease of the appendix being the primary cause of such adhesion, and the infection of the cyst.

If direct communication occur between the cyst and intestine, bladder, or vagina, suppuration results, universal adhesions occur, and the cyst may be filled with fœtid pus, and even gas, so as to be resonant. There are very severe abdominal symptoms and great wasting and exhaustion with suppurating cysts of this kind, but not necessarily a high temperature. The pus may find its way into the general peritoneal cavity or be discharged into bowel, bladder, or vagina.

Dermoids are particularly dangerous tumours,[2] as they are very liable to suppurate. They may be lodged in the pelvis for long periods, and as parturition may occur repeatedly they are liable to get bruised and inflame afterwards. They may burst and lead to the most troublesome fistulæ,[3] which may go on discharging from time to time, and sloughs, fat, hair, pieces of bone, or teeth may come away. They may give rise to great suffering if they rupture into the bladder, though cystitis does not necessarily occur. They may even be inverted into the bladder, bowel, or vagina, and thus partially expelled.

Dermoid cysts may also empty their contents through the abdominal wall, and this may lead to their cure.

Twisting of the Pedicle.[4]— Axial rotation of pedunculated tumours of the ovary is a subject of great interest and importance, as it gives rise to serious pathological changes, such as internal hæmorrhage, peritonitis, and exhaustion from suppuration. It occurs in about eight to ten per cent. of cases of ovarian and parovarian tumours. It may be gradual or acute. Various theories have been advanced as to its cause, such as sudden movement or exertion, the passage of fæces down to the rectum, or the filling and emptying of the bladder. Pregnancy seems to be an important factor in causing such rotation. Thornton records fourteen out of fifty-seven cases of axial rotation which were associated with pregnancy.[1] The rotation of the cyst in early pregnancy is probably due to the gradual enlargement of the uterus, but it may occur later, or even be deferred till after delivery as the uterus is sinking.

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[1] Bland-Sutton, *Surgical Diseases of the Ovaries &c.* p. 127.

[2] Herman, 'Suppurating Dermoids,' *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 254; *id. Diseases of Women*, p. 754.

[3] Barnes, *St. George's Hospital Reports*, vol. viii.

[4] Thornton, *American Journal of the Medical Sciences*, vol. xcvi. 1888; Herman, *Diseases of Women*, p. 754; Bland-Sutton, *Surgical Diseases of the Ovaries &c.* 1891, p. 135; Olshausen, *Die Krankheiten der Ovarien*, 1886; Lawson Tait, 'Axial Rotation of Ovarian Tumours,' *Trans. Obstet. Soc. Lond.* vol. xxii. 1880, p. 86, and discussion.

The clinical results[2] of twisting of the pedicle are very definite. It may cause rupture or sloughing of the tumour through obstruction to the vessels of the pedicle, absolute atrophy of the tumour, and even detachment of the tumour from its pedicle and the subsequent nourishment of its tissues through vascular adhesions. True gangrene is rare, but cases have been reported by Spencer Wells, Thornton, and Tait. A cyst with a twisted pedicle[3] is a very dangerous condition. Such cysts become dark brown, or even black, and generally contract adhesions to everything in their neighbourhood. Sometimes the fluid is brownish, reddish, or black, from hæmorrhage; the obstruction to the vascular supply seems to affect the veins of the pedicle first, and the arteries later; a sort of back-pressure follows, and many small vessels on the inner wall of the cyst rupture. Profuse bleeding,[4] causing marked anæmia, may occur into the cyst and tissues of cyst-wall, or even into the abdominal cavity if the cyst ruptures. Sometimes all the vessels of such a cyst are found thrombosed, and can be seen as cords full of clot. Such 'dead' cysts are very liable to further infective processes or necrosis. If all dark-coloured cysts were really gangrenous, general infection of the peritoneum and death would occur in these cases, but it is very rare.

Although the twist in the pedicle is extreme, vascular adhesions may be sufficient to nourish the cyst-wall, and in fact the cyst may be detached from the pedicle altogether, and be found in curious positions in the abdomen, anchored by adhesions which now feed the tumour. Dermoid tumours are frequently associated with twisted pedicle, Doran records several cases.[1]

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[1] Bland-Sutton, *op. cit.* p. 135.

[2] Doran, *Diseases of Ovaries*, 1884, p. 119.

[3] Lawson Tait, *Diseases of the Ovaries*, 1883, p. 295.

[4] Spencer Wells, *Ovarian and Uterine Tumours*, 1882, p. 64.

The pedicle may be twisted only slightly, or there may be many turns. The condition of thrombosis extends to the place at which the twist occurs, but on pulling up the cyst, the pedicle beyond this point is quite healthy. If the torsion is acute the symptoms are sudden and severe, and closely resemble an attack of intestinal obstruction, accompanied by sudden violent pain, distension, and vomiting. These, occurring in a patient known to have an ovarian cyst, would lead to the suspicion of sudden twisting of the pedicle, while anæmia, pain, and sudden increase in the size of the cyst would indicate bleeding into its interior. On the other hand, if the twisting be slow and chronic, no severe symptoms occur, adhesions gradually form, and, as before explained, the cyst may live. Such tumours are generally small and are often dermoid, but it occurs with all varieties[2] of ovarian and parovarian cysts, and also with solid tumours. Large tumours are more liable to acute torsion than small ones.[3]

Rupture of Ovarian Cysts.—Rupture of a cyst,[4] as a rule, consists in mere leakage from a thin-walled loculus, and generally produces but trifling symptoms. Much, of course, depends on the character of such a cyst and its contents, for a malignant cyst may rupture and be followed by dissemination of new growth in the abdomen, or there may be true acute rupture[5] of an innocent ovarian cyst, followed by fatal hæmorrhage and peritonitis. In such cases there may have been previous twisting of the pedicle, followed by thrombosis of vessels, intracystic hæmorrhage and necrosis.

Sometimes the most puzzling conditions are met with in burst cysts. The abdomen may be full of ovarian fluid, while in the pelvis is a mass which may be diagnosed as malignant. The writer assisted Meredith to remove a tumour, in which several exploratory operations and repeated tappings had been undertaken, and its removal considered impossible; but it turned out to be a burst papillomatous cyst and was removed without difficulty. The specimen is in St. Bartholomew's Hospital Museum.[1]

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[1] Doran, *op. cit.* p. 122; specimens Royal College of Surgeons Museum, Nos. 4549–4551.

[2] Bland-Sutton, *op. cit.* p. 142.

[3] Acute and chronic torsion has been observed in the same patient (Doran, *Brit. Med. Journ.* vol. ii. 1900, p. 75).

[4] Meredith, *Trans. Path. Soc.* vol. xxxi. p. 180.

[5] Doran, *op. cit.* p. 105.

Meredith[2] reports a case which had ruptured spontaneously thirty-four times in nine years; finally the cyst was removed by operation. Each rupture was accompanied by sudden pain, and the tumour altered its shape. Profuse diuresis followed each bursting, but the fluid gradually re-collected.

Another remarkable case is quoted by Bland-Sutton[3] in which a woman was tapped eighty times, and over 6,000 pints of fluid altogether removed, in a period of 26 years.

Rupture of ovarian cysts into the bowel, vagina, or bladder has already been mentioned in considering inflamed cysts, especially dermoids. If adhesions occur to bowel it is generally the sigmoid flexure or rectum; if intestinal gases enter suppuration occurs, and pus finds its way into the bowel.[4] Ovarian tumours have been known to burst into the stomach, or small intestine, or through the abdominal wall, or an ovarian cyst may become adherent to the Fallopian tube and burst into it, forming a tubo-ovarian cyst. True ulceration[5] of the wall of an ovarian cyst, leading to rupture, does not occur; the so-called ulcers are atrophic patches in the wall of a secondary cyst which is undergoing degeneration.

Direct violence occasionally may rupture an ovarian cyst, or it may burst during an ordinary abdominal examination, or during delivery, the rupture generally taking place at some weak spot in the cyst-wall.

Myxoma of the Peritoneum.—In this condition, which is very rare, the peritoneal cavity contains masses of jelly-like myxomatous tissue and fluid. The omentum and the whole peritoneum, including the serous coat of the intestines, are thickened and covered with myxomatous masses. Whether this condition be a distinct peritoneal growth or a diffuse degeneration is not quite certain; 'myxoma,' 'pseudo-myxoma,' 'peritonitis pseudo-myxomatosa,' are names given to it.[6] The myxomatous tissue increases rapidly and the fluid is very thick. There may be adhesions. Nothing is known of its pathology. It occurs chiefly in multiparæ between 40 and 60.

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[1] St. Bartholomew's Hospital Museum, No. 2913 D.

[2] Meredith, *Trans. Path. Soc.* vol. xxxi. p. 180; and Doran, *op. cit.* p. 117.

[3] Bland-Sutton, *Surgical Diseases of the Ovaries and Fallopian Tubes*, new ed. 1896, p. 133 (the specimen is in the Museum of the Royal College of Surgeons); *id. Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 98 (colloid cyst).

[4] Herman, *Diseases of Women*, p. 759.

[5] Doran, *op. cit.* p. 116.

[6] Herman, *op. cit.* p. 751.

Many cases of myxoma have followed the rupture of ovarian cysts, and it seems probable when it occurs after the surgical removal of a cyst that some part of the original tumour has been left behind. This is especially likely to occur in papillomatous cysts.

There is no proof that it is a new growth of the peritoneum itself.

Pressure Effects.—Large tumours extending upwards into the abdomen may cause great distension. Skin-cracks may occur, as in pregnancy, also enlargement of parietal veins, and displacement of abdominal viscera; or, if the tumour encroach upon the cavity of the thorax, it may push the lower ribs outwards, and interfere with the functions of the heart and lungs.

In the pelvis, veins may be compressed, leading to œdema of the lower limbs; fixed tumours may even press on the inferior vena cava. The rectum, bladder, and urethra are sometimes interfered with in a similar way.

Kidneys, Ureters.—Very serious complications may arise in connection with the kidneys and ureters.[1] There may be mechanical interference with the escape of urine, and hydronephrosis, or inflammatory changes originating in the bladder. Great care should be taken to examine the amount of urine and its constituents, in cases of ovarian tumour, as serious renal troubles may already be in progress, calling for immediate removal of such a growth. Diseased kidneys may unfortunately be the cause of death after operation.

The serious septic affections connected with the kidneys in such cases probably arise from cystitis, which spreads upwards, giving rise to suppurative nephritis and pyonephrosis.

Intestinal obstruction caused by ovarian cysts is not common. It is generally due to a piece of gut becoming strangulated round the pedicle of a cyst, or round some firm adhesion, rather than by mere pressure from the size of the cyst. It has also occurred from implication by the growth, after rupture of a papillomatous cyst.

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[1] Bland-Sutton, *op. cit.* new ed., 1896, p. 137; and Doran, *Tumours of the Ovary, &c.*, 1884, p. 155

Pregnancy and Labour, complicated by Ovarian Cysts.—Any kind of ovarian tumour may complicate pregnancy, and the disease may be bilateral. Ovarian cysts associated with pregnancy have already been referred to, and their dangers pointed out, for the tumour itself is a constant source of danger. Dermoids[1] are most frequently found complicating pregnancy, as they more usually occur in young subjects, who are the more likely to become pregnant. Dermoids grow slowly and pregnancy may occur at any time during their growth.

Labour[2] may be complicated by the presence of an ovarian tumour either in the abdomen or pelvis. Such a tumour seems to do but little harm so long as it is above the pelvic brim, unless it be very large. It is liable to become inflamed after delivery. In the pelvis it may get jammed down in front of the fœtal head, or may prevent its descent if growing in the broad ligament. Cases are recorded[3] where cysts have been driven down by the advancing head and have burst through the posterior vaginal wall. Griffith[4] has reported a case where a solid tumour of the ovary (fibroma) obstructed labour and was thought during life to be the head of another fœtus which was extra-uterine. One half of the specimen is in St. Bartholmew's Hospital Museum[5] (see fig. 105), the other is in the Museum of the Royal College of Surgeons.

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[1] Herman, *op. cit.* p. 763.

[2] McKerron, 'The Obstruction of Labour by Ovarian Tumours in the Pelvis,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 334.

[3] Griffith, Allbutt and Playfair, p. 852; and Olshausen, *op. cit.*

[4] Griffith, 'A Case of Obstructed Labour in which a large Fibroma of the Ovary occupying the Pelvis was mistaken for the Head of an Extra-uterine Fœtus,' *Trans. Obstet. Soc. Lond.* vol. xxxiii. 1891, p. 140.

[5] St. Bartholomew's Hospital Museum, 3091 A.

CHAPTER XV.

ON CANCER OF THE UTERUS.

No subject is perhaps more interesting, both to practitioner and pathologist. The frequency with which it occurs, its terrible results, and its fatal end, alike demand both careful attention and study. And yet perhaps no subject is more difficult and confusing, both clinically and pathologically, so varied are the types of malignant disease met with at the bedside, and still more so their pathological appearances under the microscope. Yet both clinical and microscopical evidences of malignant disease are all-important ; the one alone may be sufficient, but together they may form the solution of a problem of exceeding difficulty.

The microscopical appearances of uterine neoplasms, and their differentiation into carcinoma, sarcoma, or adenoma, are very puzzling. So varied are their appearances, even in the same section, that it requires a considerable knowledge of uterine histology before an opinion can be arrived at. Even then varied views may be taken, as it is possible that the malignant diseases, which we call epithelioma, carcinoma, and sarcoma, may all be present in the same individual, and that in attempting any classification of malignant disease of the uterus we do so with regard to the most striking forms met with, and only with anything like precision in comparatively early stages of the disease. The terms used simply indicate certain pathological tendencies, and imply that the ultimate facts, determining their origin and their relationships, are still quite unknown to the pathologist.

The varieties of malignant disease which affect the uterus are Carcinoma, Sarcoma, and Adenoma. Sarcoma is a growth of connective-tissue type; it originates from connective tissue and grows from its constituent cells. Carcinoma and adenoma contain epithelial elements, and a stroma in which such epithelial elements are embedded. Some of these are built exactly on the plan of glands, and the new-formed tissue (adenoma) corresponds to a definite glandular type. In carcinoma there is no such orderly arrangement, by the multiplication of cells or glands, epithelium and fibrous tissue are intermingled, and we have clusters of cells embedded in connective tissue whose elements are likewise multiplying. Yet the distinction is often very difficult, and growths of a connective-tissue type may assume an alveolar form, the cells may be indistinguishable from epithelial cells, and on anatomical evidence alone a sarcoma may be taken for a carcinoma, or *vice versa*.

Little or nothing is known of the **cause** of malignant disease generally, still less so of its causes when it starts in the uterus itself. The following theories regarding the origin of New Growths have been adduced, but it is very doubtful if any one of them throws very much light on the subject.

(*a*) **The Inclusion Theory, or the Embryonic Hypothesis of Cohnheim.**[1]—According to this theory, tumours are developed from embryonic cells which, having been produced in greater numbers than are required for the formation of fœtal tissues, still remain in the locality in which they grew, and later, either by normal physiological action or artificial irritation are lighted up into active growth, forming a neoplasm.

(*b*) **Hereditary Influence.**—More will be said of this, but because there is a clear family history of similar growths in a certain number of cases, it does not prove that tumours are of constitutional origin, for they may be inherited as local affections, just as club-foot or other deformities (Bowlby).

(*c*) **Irritation and Injury.**—As the matter stands at present, we can only say that 'while fœtal inclusion and hereditary taint may act as predisposing causes of the origin of tumours generally, irritation and injury may be considered as exciting causes.' It appears from clinical evidence that long-continued irritation is necessary for the production of epitheliomatous tumours, but that the occurrence of sarcomata is influenced rather by direct and sudden injury.[2]

The question is considered later in connection with the uterine neoplasms, as to the possibility of origin from the continued irritation of childbirth, or from lacerations.

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[1] Bowlby, 'On the Causes of Tumours,' *Surgical Pathology*, 4th ed. p. 158 *et seq.*

[2] Bowlby, *op. cit.* p. 159.

(*d*) **Physiological Activity and Decline.**—Sarcomata are prone to develop in early life in rapidly growing connective tissue, whilst carcinomata grow in glands and other structures undergoing retrograde changes, such as the breast or uterus after the child-bearing period.[1]

Parasites in Cancer.—Much attention of late years has been devoted this subject and some observers are of the opinion that protozoa can be demonstrated in the cells of carcinoma and sarcoma.

Most pathologists consider that such bodies are not parasitic, and attribute them to cell inclusions or degenerations of cell protoplasm (Bowlby). Recent observers [2] class the parasite under the heading of a fungus (blastomyces), or as a protozoon. Attempts at cultivation of protozoa in cancer have been recently successful.

More will be said of this in the chapter on the Relations of Micro-organisms to the some of the Diseases of Women.

At present it seems safe to say that there is no *direct* evidence that tumours are caused by parasitic infections.[3]

As to the origin of cancer of the uterus, whether the ultimate fact be some change in the connective tissue alone or in the epithelial cells, we do not yet know. Virchow probably first propounded the view of the connective-tissue origin of cancer of the cervix, while Waldeyer and Klebs support the epithelial origin. Ruge and Veit [4] maintain that the pavement epithelium of the portio is never the origin of epithelioma or cancer of the vaginal portion, but that the starting point is either in the deeper layers of the connective tissue, or in the newly-formed glands of erosions, and that the connective-tissue stroma becomes vascularised and passes into the embryonic condition, the new cellules assuming an epithelioid aspect.[5]

English writers mostly, and perhaps rightly so, follow the epithelial origin of cancer. Sir John Williams[1] defines cancer anatomically and genetically, as an alveolar new growth, containing epithelial structures which have originated in previously existing epithelium.

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[1] Bowlby, *op. cit.* p. 159.

[2] Mitchell Banks, 'Lettsomian Lectures on Cancer of the Breast,' *Lancet*, vol. i. 1900, pp. 684, 843, 990, with plate. 'Etiology of Cancer,' *Lancet*, vol. i. 1901, p. 1413; Gaylord, 'Protozoon of Cancer,' *Amer. Jour. of Med. Sciences*, May 1901. See also chapter on 'Micro-organisms in relation to Diseases of Women,' in this book, with appendix.

[3] Bowlby, *op. cit.* p. 160. See also Kanthack, *ibid.* p. 13.

[4] Ruge and Veit, *Zeit. für Geb. und Gynäk.* 1881.

[5] Sinclair, Allbutt and Playfair, p. 650,

Anatomy and Histology.—The various parts of the uterus and their epithelium must be studied in detail in relation to the origin and spread of cancer.

The uterus consists of **body** and **cervix**, and the cervix itself consists of a vaginal portion and a supra-vaginal portion. The vaginal portion protrudes into the vaginal cavity, the supravaginal extending above this to the isthmus (internal os), where it joins the body of the uterus proper.

The **vaginal portion** is covered with squamous epithelium which ends at the external os.[2] At this point a zone of transitional epithelium occurs, before the typical columnar epithelium of the canal is reached.

The transitional epithelium is a narrow and debatable belt of cubical cells, with a few glands, which is said to belong neither to the lining membrane of the canal nor to the squamous epithelium of the vaginal portion. Sir John Williams classes it with that belonging to the epithelium covering the vaginal portion, and gives the inner end of the line limiting the portio vaginalis above as being a little within the cervical canal.

A similar debatable boundary-line is described at the upper termination of the cervical canal, as it passes the constriction of the internal os, to become the body proper. This is the spot where malignant ulceration is for a time checked when spreading upwards from the cervix.

The interest of such transitional areas in other regions of the body is of importance in the question of the origin of neoplasms.

The **cervix proper** is chiefly characterised by containing glandular elements of great complexity and size, lined with tall columnar epithelium,[3] which are reduplications of the surface epithelium. Above this (*i.e.* above the internal os), the tubular glands characteristic of the body of the uterus begin.

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[1] *Cancer of the Uterus*, p. 5; Harveian Lectures, 1886.

[2] Cullen, *Cancer of the Uterus*, 1900, pp. 17–18, states that the upper limit of the squamous epithelium may extend upwards into the cervical canal for some distance, and that its limits are subject to much variation.

[3] 'In the cylindrical epithelium of the cervix the cell is relatively very high being nearly twice as high as that of the body, but much more slender' (Cullen, *op. cit.* p. 18).

This division of the uterus into various boundaries is of some importance, because cancer may begin in any one of them, but **cancer of the cervix** as a whole, and **cancer of the body,** appear to be the two great varieties, and these will be described in the present chapter. As regards the question of making a hard and fast distinction between the vaginal portion and cervix, the writer does not propose to do so. Such a distinction is an arbitrary one[1] but there is certainly a great difference between cervix and body, not only in structure and physiological action, but in the new growths which arise from each.

Classification of Cancer of the Uterus.

a. **Cancer of the Cervix.**

1. Squamous-celled Epithelioma of vaginal portion.
2. Glandular or Columnar-celled Carcinoma of cervix proper. [Adenoma malignum of the cervix and sarcoma are very rare.]

b. **Cancer of the Body.**

1. Carcinoma.
2. Sarcoma. (Including Deciduoma malignum.)
3. Adenoma malignum.

Epithelioma of Vaginal Portion.—This begins in the stratified epithelium on the vaginal surface, or in the zone of transitional epithelium at the os externum. By cancer of the vaginal portion is meant cancer starting there, not cancer which has spread down and involved the vaginal portion. This is common enough, and indeed, in the vast majority of cases seen, it is impossible at the time to say in what portion of the cervix such a growth may have originally started.

Sir John Williams,[2] in the Harveian Lectures for 1886, which embody the views held by most English gynæcologists at the present time and form a classical survey of the whole subject, says that 'cancer may begin on any point of the vaginal portion; it may even begin at more than one point, or it may originate in the external orifice.'

In its early stages (fig. 114) cancer of the vaginal portion

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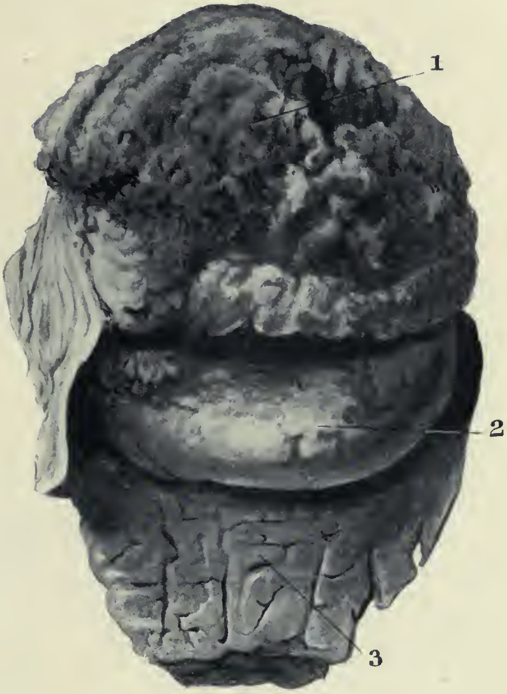
[1] J. Sinclair, Allbutt and Playfair, p. 653.

[2] *Cancer of the Uterus*, p. 29.

Fig. 114.

CANCER OF THE CERVIX. (Early Stage.)



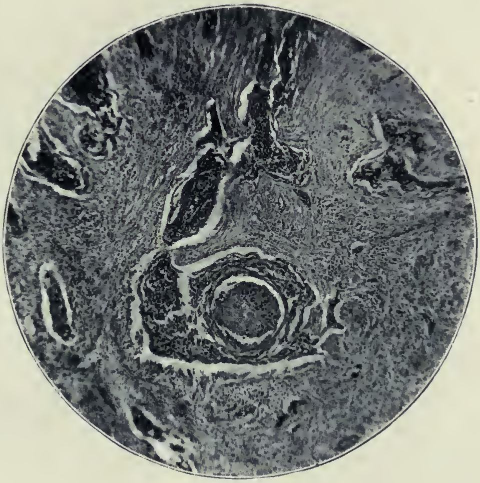
Portion of the cervix uteri and vaginal wall. The anterior lip shows an early stage of cancer; it is enlarged, and a raised friable mass of growth is seen springing from its surface. The posterior lip is free from growth except at the extreme left-hand corner. As no microscopical examination has been made, it is impossible to say whether the growth is an epithelioma or a glandular cancer.

1. Mass of raised soft cancerous growth springing from the anterior lip of the cervix.
2. Posterior lip.
3. Portion of vaginal wall.

(Specimen, W. H. Tate. Museum, St. Thomas's Hospital.)

Fig. 115.

EPITHELIOMA OF THE CERVIX.



Section of a portion of the cervix uteri which was the seat of a cancerous growth. The tissues are invaded from above downwards by a well-marked column of squamous epithelial cells. In the centre of the specimen is a well-defined cell-nest, and grouped around it are areas of epithelial cells more or less flattened by pressure. Other similar columns of cells are seen invading the tissues at the top left-hand side of the section.

(Microphotograph by C. H. Cosens.)

assumes no special form; there may be a red tuberculated area or an uneven surface with a granular appearance, or the cervix is red, angry looking, and ulcerated.[1] Sometimes it may take the form of a friable papillary growth with out-turned edges, or the disease may appear as a nodule or nodules, just at, or within, the os externum.[2] Terms which have long been used, such as 'mushroom-shaped variety' and 'cauliflower excrescence,' should be given up; they are confusing and mean nothing; 'it matters nothing what vegetable a cancer happens to resemble.'[3] There is a true cauliflower excrescence; it is a form of cancer of the vaginal portion and consists of a soft fringed growth having a stalk; it is very rare. Sir John Williams describes such a case, and reports it as a squamous-celled epithelioma, which was villous and soft.

In examining sections of maligant disease of the vaginal portion, the type is almost invariably a *squamous-celled epithelioma*, and, as the area of disease is approached from the normal vaginal epithelium, it is noticed that the deeper layers become thicker, and these thickened layers of squamous cells are thrown into folds by the papillæ growing from beneath. These processes in the diseased parts become very long and irregular as they advance towards the surface of the ulcer, where the stratified epithelium is practically lost. Surrounding the processes of these papillæ there is everywhere a dense mass of stratified epithelium whose deeper layers are in a state of intense activity,[4] strings of these cells running down into the tissues beneath (fig. 115); they are of all shapes and sizes, but in the main keep their original epithelial form,[5] and can be easily recognised. Typical cell-nests occur only occasionally (fig. 115). These processes penetrate everywhere deeply into the cervical tissues, and in microscopical sections are puzzling if cut in cross section, as they appear as rounded islands of cells, which at first sight seem as if they were contained in alveoli, thus imitating carcinoma. Sometimes, if the disease be near the os externum, true glands may be met with like those in an erosion, some healthy, some crowded among the epithelial cell processes, others themselves filled with cancerous growth actually growing into their lumen. Sometimes epithelial cells are found more or less free in the stroma, almost imitating sarcoma, but differing from the surrounding stroma by their epithelial-like shape and large nuclei. Cell-nests are not often met with in these advanced cases. There is nearly always an active infiltration of small cells into the surrounding stroma. Generally such epitheliomatous patches on the cervix are more or less superficial, but the proliferation may go on to such an extent that a large mass of growth is found filling the whole vagina (fig. 116), which easily breaks down under the finger ; such a growth may have a narrow base. On section the stroma is scanty, the cell masses constitute almost the whole growth, having a few delicate trabeculæ among them which carry the vessels, the connective tissue itself being frequently of an embryonic character. Sometimes the invasion of the cervix at the base of such a tumour is very slight indeed, sections show little or no infiltration, and in many cases after removal of such growths no return has taken place, showing the superficial character of the disease.[1] Some of the large masses found in the vagina growing from the cervix are probably of the nature of papillary epitheliomata,[2] and the cervix, though invaded, remains movable, the cellular tissue being involved only very late in the disease.

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[1] Sir J. Williams, *Cancer of the Uterus*, p. 25.

[2] Cullen, *Cancer of the Uterus*, pp. 41–45, traces the following stages in squamous-celled carcinoma of the cervix:

1. Hardness and induration of cervix without loss of tissue.
2. Moderate disintegration of the cervix.
3. Extensive or complete involvement of the cervix.

[3] Herman, *Diseases of Women*, p. 356.

[4] Cullen, *Cancer of the Uterus*, 1900, p. 47.

[5] See Delbet, article on 'Cancer of the Uterus,' *Traité de Chirurgie*, 2nd ed. vol. viii. p. 191. For changes in the mucosa of the body of the uterus in cancer of the cervix, see Abel, *Arch. f. Gyn.* vol. xxxii. p. 2; Fränkel, *ibid.* vol. xxxiii. p. 1; Marchand, *Soc. de Chir.* January 13, 1892.

Sir John Williams reports two curious cases, one in which cancerous changes had commenced in a mucous polypus,[3] and another in which a fibroid polypus was the seat of a superficial epithelioma of a typically squamous character. Sir John Williams says that he has never seen this disease start from a tear of the cervix, nor is there any evidence that ulceration of the cervix plays any part whatever in the etiology of this form of cancer.[4]

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[1] Lewers, 'After-Results of Vaginal Hysterectomy,' *Lancet*, vol. i. 1901, p. 9.

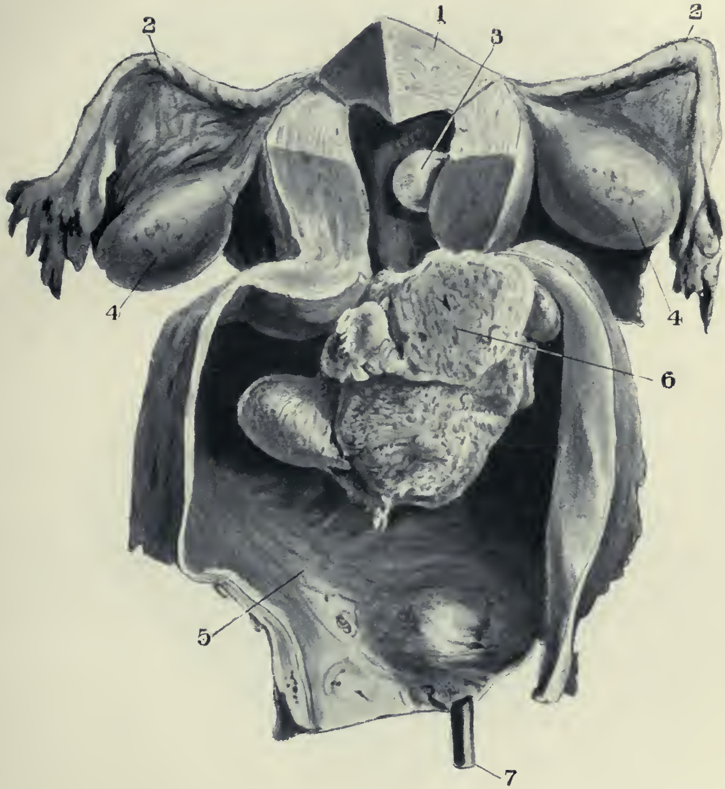
[2] Their histology, however, is very puzzling.—Author.

[3] *Cancer of the Uterus*, pp. 29, 72.

[4] *Ibid.* p. 31.

Fig. 116.

EPITHELIOMA OF THE CERVIX.



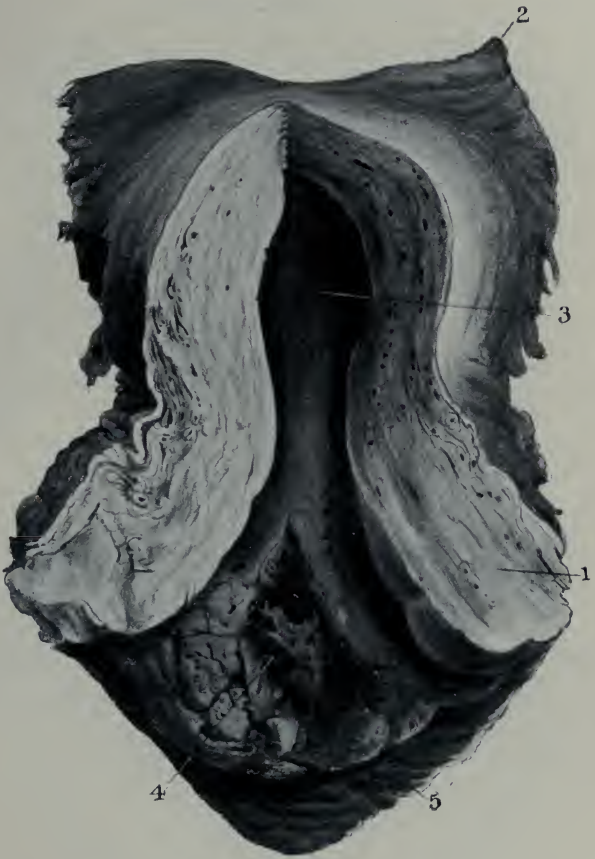
A uterus and vagina with Fallopian tubes and ovaries. The vagina is occupied by a large soft sprouting growth springing from the cervix uteri. A small mucous polypus is seen in the body of the uterus.

1. Fundus uteri.
2, 2. Fallopian tubes.
3. Mucous polypus.
4, 4. Ovaries.
5. Vagina.
6. Epitheliomatous growth springing from the cervix uteri.
7. Glass rod placed in the urethra.

(Specimen 4661. Museum, Royal College of Surgeons.)

Fig. 117.

CANCER OF THE CERVIX. (Early Stage.)



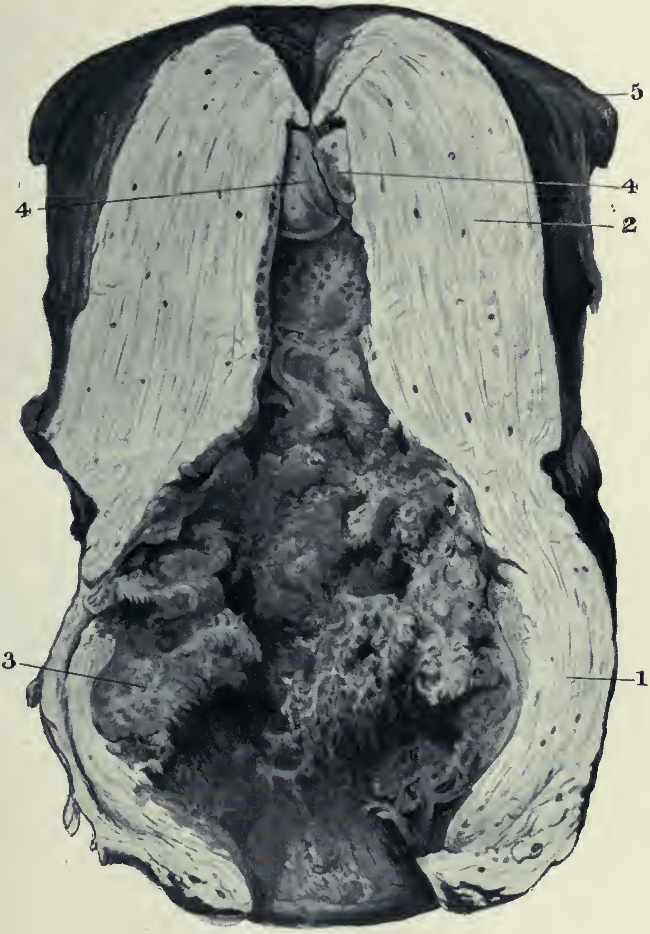
A uterus laid open from behind, showing an ulcer on the anterior wall of the cervix. It has a raised edge, and the surface is covered with shreds of sloughing tissue. Microscopically it is a columnar-celled carcinoma. It was removed by vaginal hysterectomy from a woman of 54.

1. Posterior portion of the cervix.
2. Fallopian tube.
3. Cavity of body of the uterus.
4. Surface of cancerous ulcer on anterior wall of the cervix.
5. Vagina.

(Specimen 4664 c. Museum, Royal College of Surgeons.)

FIG. 118.

CANCER OF THE CERVIX.



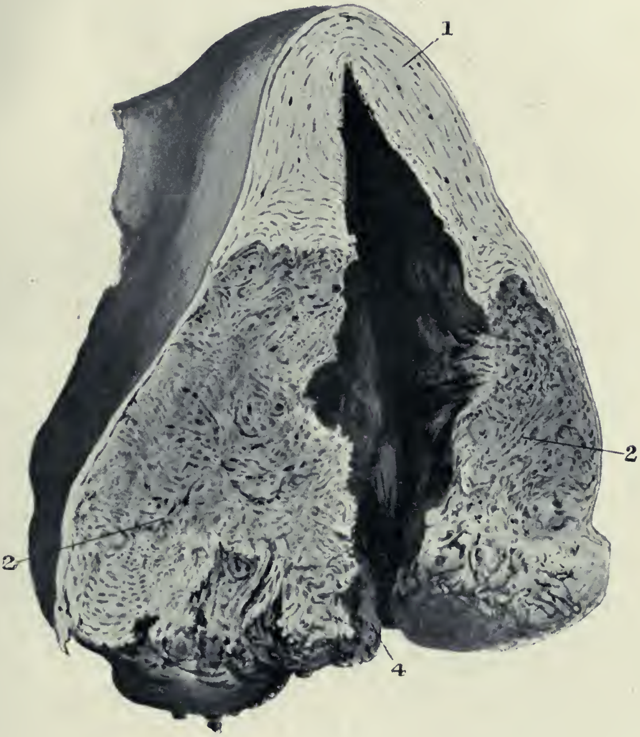
A uterus in which the cavity of the cervix is occupied by a soft papillary growth Microscopically it is a carcinoma.

1. The much-expanded cervix.
2. Fundus uteri, which is free from growth.
3. Portion of friable papillary carcinomatous growth
4, 4. Simple mucous polypi.
5. Fallopian tube.

(Specimen, W. H. Tate, No. 12. Museum, St. Thomas's Hospital.)

Fig. 119.

CANCER OF THE CERVIX (ADVANCED).



A uterus in which the lower two-thirds of the walls are enlarged by the infiltration of a soft cancerous growth. The cervix is much expanded, and the lower surface of the growth is ulcerated and flocculent.

The growth is spreading up into the body of the uterus. It is a columnar-celled carcinoma, and probably began in the glands of the cervix.

1. Body of uterus.
2, 2. Masses of cancerous growth involving the cervix, which is much enlarged below.
4. Region of external os; here the growth is breaking down.

(Specimen 3008. Museum, St. Bartholomew's Hospital. See also Sir John Williams, 'Cancer of the Uterus,' plate xiii. fig. 1.)

Thus epithelioma of the vaginal portion is generally a superficial growth,[1] and its direction of extension is not towards the cavity of the uterus, but outwards and downwards as a large mass into the vagina and along its walls. It does not tend to spread to the cellular tissue, or, at all events, not till very late in the disease; and as it is situated in a part that can be seen and felt, it should easily be found out, and offers, of all forms of cancer of the uterus, the most hopeful prospect for treatment.[2]

This form appears much more frequently during menstrual life than after the menopause. Pregnancy seems to have little to do with it. Hæmorrhage is the early symptom and fœtid discharge may be present in the earlier stages (Williams).

Columnar-celled Cancer of the Cervix proper (Adeno-carcinoma).—This form begins in the columnar epithelium of the canal or more commonly the glands of the cervix. It is more common and more malignant than epithelioma. It may begin low down in the cervix—indeed, this is far commoner [3] than the form which starts near the internal os but it may begin at any part,[4] it may co-exist with an erosion, and is said to be more common on the posterior lip. The forms assumed by this variety are varied.[5] It may occur as a nodule in the wall of the cervix (fig. 117) or as enlargement of one lip which soon breaks down. Sometimes the growth begins high up in the cervical glands, and there may be nothing to show, or perhaps the cervix feels only a little enlarged and to the touch almost natural, as the growth is still covered and concealed by the remains of normal cervical tissue.[6] Sooner or later this is involved and breaks down, and the whole cervix is found to be hollowed out and deeply involved (figs. 118, 119), a large funnel-shaped excavated ulcer with irregular edges now occupying its position (figs. 119, 120). The so-called 'mushroom growth' arises from hypertrophy of the parenchyma of the cervix, which is infiltrated with cancerous elements; it is almost always a carcinoma. Columnar-celled cancer of the cervix, in whatever position it may start, tends to grow **in all directions**, as well as upwards and downwards. It involves the whole thickness of the cervix, and infects the cellular tissues around the cervix early, hence it is the most unfavourable form of uterine cancer. Indeed, in cases which start high up in the cervix the cellular tissues are involved, and the disease has already made hopeless progress long before it can be detected by any vaginal examination.

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[1] Delbet, *Traité de Chirurgie*, 2nd ed. vol. viii. pp. 189–191, distinguishes the varieties as—(1) 'Forme papillaire'; (2) 'Forme interstitielle.' Delbet thinks that epithelioma of the vaginal portion of the cervix and epithelioma of the vagina are identical (p. 199). On 'The Extension of Cancer of the Cervix,' see Bigeard, *Thèse de Paris*, 1899.

[2] Griffith, 'Early Diagnosis of Cancer of Cervix Uteri,' *Brit. Med. Journ.* vol. i. 1896, p. 265.

[3] Cullen, *Cancer of the Uterus*, 1900, p. 40, states that squamous-celled carcinoma is the most common form of cancer of the uterus.

[4] Waldeyer, *Die Entwickelung der Carcinome*; *Virchow's Archiv*, 1872, vol. lv.

[5] Sir J. Williams, *Cancer of the Uterus*, p. 74.

[6] Cullen, *Cancer of the Uterus*, 1900, p. 281.

The chief type of cancer affecting the cervix appears to be a **columnar-celled glandular carcinoma**, but many variations are seen. Sections of such a growth show gland-like spaces[1] (fig. 121), some lined with a single layer of cells, others with two or more layers, and others in which there is no lumen whatever. Sometimes masses of epithelium project from the lumen and run riot among the surrounding stroma (fig. 122), as in mammary cancer. Usually the cancerous cells are columnar, but cells may occur differing very widely from normal cervical epithelium. In the large alveolar spaces these are easily detached. The cells multiply by indirect nuclear division, and the nuclei of cancer cells are large, abundant in chromatin, staining readily, especially along the advancing margins of the growth, and often originate karyokinetic figures.[2] Asymmetrical and multipolar mitoses are described as taking place frequently in cancer cells. Often more than one nucleus is present, and their chromatin tends to be shed into the surrounding protoplasm before division. These appearances indicate marked reproductive activity.[3] Cancer cells may also undergo mucoid, caseous, fatty, or calcareous degeneration. The connective-tissue stroma of the new growth is mainly cellular, often showing myxomatous changes. As before

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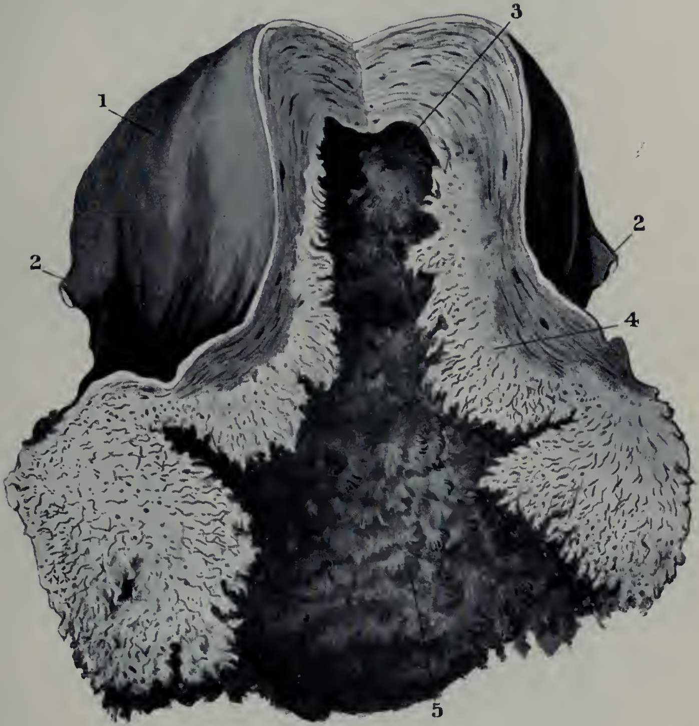
[1] Delbet, *Traité de Chirurgie*, 2nd ed. vol. viii. p. 196, calls them cylindrical epitheliomata, and believes they start in the glands. See also Ruge and Veit, 'Der Krebs der Gebärmutter,' *Zeit. für Geb. und Gynäk.* 1881, vol. vi. p. 281, 'on the origin of carcinoma from the cervical glands.'

[2] Roger Williams, quoted by Clarence Webster, *Diseases of Women*, p. 587.

[3] Cullen, *Cancer of the Uterus*. 1900, p. 283; also *ibid.* p. 655, 'Methods of Cell Multiplication in Cancer.'

Fig. 120.

ADVANCED CANCER OF THE CERVIX.



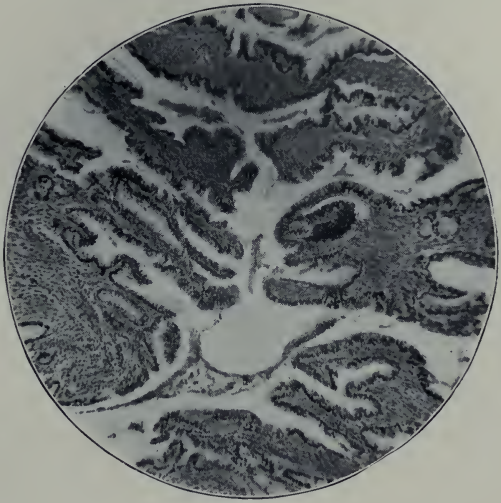
A uterus and portion of the vagina to show the results of cancer beginning in the cervix. The growth has spread to the body of the uterus, and the vagina is everywhere involved. The whole growth is undergoing necrotic changes. Microscopically it is a columnar-celled carcinoma.

1. Body of the uterus.
2, 2. Fallopian tubes.
3. Cavity of the body of the uterus, to which the growth has already spread.
4. Region of the internal os.
5. Vagina and remains of cervix involved by a soft flocculent necrotic cancerous growth.

(Specimen 3009. Museum, St. Bartholomew's Hospital.)

Fig. 121.

COLUMNAR-CELLED CARCINOMA OF THE CERVIX.

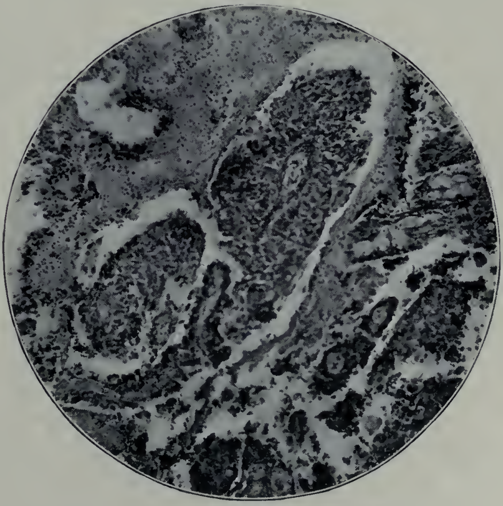


Section of a cancerous growth which deeply involved the cervix uteri. The tissues are everywhere invaded and occupied by tracts of atypical columnar cells arranged in irregular acini. The cells themselves have undergone much proliferation, but the growth has more or less preserved its glandular type. In places the cells have fallen out during the processes of hardening.

(Microphotograph by C. H. Cosens.)

Fig. 122.

ADVANCED CANCER OF THE CERVIX.



Section of a cancerous growth which had practically destroyed the cervix, and had extended as far as the os internum. It shows large masses of atypical columnar epithelial cells undergoing rapid proliferation, which have infiltrated the tissues in all directions. Very little of the normal stroma of cervix remains. The growth is very vascular, and there are hæmorrhages in places. It is a columnar-celled carcinoma. The body of the uterus was occupied by a soft growth which was also thought to be cancerous, but on section was a simple adenoma.

(Roberts, 'Trans. Obstet. Soc. Lond.' vol. xlii. 1900, p. 267. Microphotograph by C. H. Cosens.)

stated, great care must be taken in examining sections[1] to avoid errors of interpretation. Sections of normal glands cut obliquely look like cancerous alveoli; serial sections alone can decide.[2] The arrangement of cells is important: regular and orderly in normal conditions, irregular in shape, size, and arrangement in cancer.

The greatest difficulty is sometimes experienced, in examining sections of cancer of the cervix, in deciding whether the type of cancer cells found be glandular or not. Sometimes the alveolar form is preserved, in others there is no gland-like formation, the cells being cemented together in masses crowding the alveolus. In other parts cells may be seen which invade the connective tissue in columns and masses not very far different from those seen in squamous epithelioma.

Carcinoma of the cervix varies considerably in hardness in proportion to the relative amount of cells or fibrous tissue which exists. Some observers even describe a form apart from the common glandular type, the 'scirrrhous' variety of cancer,[3] which is said chiefly to affect old women. It is a hard form of cancer, there is comparatively little hypertrophy of the uterus itself, the body is not invaded, there is little tendency to hæmorrhage or ulceration, but early and often hopeless invasion of the cellular tissue. For this reason, though the uterus feels movable, it is an exceedingly unfavourable form for operative procedure.

We have no means of determining exactly whether a growth felt on vaginal examination be an epithelioma or a carcinoma, and if the latter, which variety.

Clinically the types of cancer of the cervix met with on vaginal examination fall into two main groups; either—

(*a*) A hypertrophic irregular condition of the vaginal portion of the uterus, which is friable[4] and readily bleeds.

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[1] Nothing is more puzzling than sections of uterine new growths, and the varieties exhibited in microscopical preparations vary within very wide limits. —Author.

[2] Clarence Webster, *op. cit.* p. 508.

[3] W. J. Sinclair, article 'Malignant Disease of the Uterus,' Allbutt and Playfair, p. 672.

[4] For the importance of 'friability' of tissues affected with cancer, see W. J. Sinclair, *op. cit.* p. 674.

b) Or an excavation with hard irregular edges, such excavation already meaning great advance in the progress of the growth, the tissues around being often hopelessly involved.

Nothing is more important than the **early recognition of cancer.**[1] How often do we see cases in which the diagnosis is easy but all possibility of operation is hopeless ![2] It is well to remember that uterine hæmorrhage at any time should be regarded as pathological, particularly that occurring at, or after, the menopause. Such cases should always be examined. It is but too often that we find that the patient is afraid to come, or afraid of the verdict, till it is too late ; or the symptoms are discounted both by the patient and her medical adviser.

Sarcoma of the Cervix[3] is a very rare disease, and is said to occur as a papillary tumour growing from one or other lip. Spiegelberg's case in a girl of 17 was a papillary tumour of the anterior lip, which returned ten months after removal and filled the whole of the vagina. It resembled a hydatidiform mole of the chorion, but examination proved it to be a sarcoma with œdema of the stroma.

Pernice[4] describes another case in which the tumour was like a bunch of violet-coloured grapes. It was removed, but an abdominal tumour formed rapidly.

Pfannenstiel[5] has collected other cases of cervical 'cluster-sarcoma.' Drawings of such cases of sarcoma of the cervix can be found in Pozzi.[6] Most of these cases occurred in young women, and were rapidly fatal. Cartilage has been seen in one such tumour. Other varieties[7] of sarcoma, besides the grape-

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[1] Griffith, 'The Early Diagnosis of Cancer,' *Brit. Med. Journ.* vol. i. 1896, p. 264. Also Herman, *ibid.* vol. i. 1894, p. 1009 ; and leading article, 'The Early Recognition of Uterine Cancer,' *ibid.* vol. i. 1901, p. 164.

[2] Cullen, *Cancer of the Uterus*, 1900, p. 326, 'On the Organs secondarily involved.'

[3] Spiegelberg, *Arch. f. Gyn.* 1879, vol. xiv. p. 178; *ibid.* 1880, vol. xv. . 437; and vol. xvi. p. 124. Also Whitridge Williams, 'Contributions to the Histology and Histogenesis of Sarcoma of the Uterus,' *Amer. Journ. Obstetrics*, 1894, vol. xxix.

[4] Pernice, *Virchow's Archiv*, 1888, vol. cxiii. The specimen is figured in Bland-Sutton and Giles, *op. cit.* p. 205.

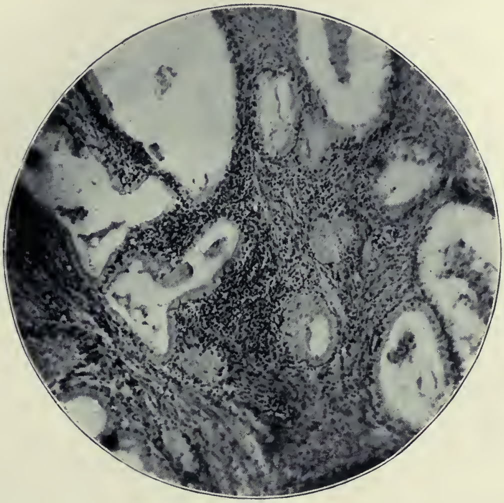
[5] Pfannenstiel, 'Das traubige Sarcom des Cervix Uteri,' *Centralb. f. Gyn.* 1891, no. 42, p. 855, and *Virchow's Archiv*, vol. cxxvii. 1892.

[6] Pozzi, New Sydenham Soc. translation, vol. ii. pp. 22, 23.

[7] Cullen, *Cancer of the Uterus*, 1900, p. 199.

Fig. 123.

CYSTIC ADENOMA OF THE CERVIX.

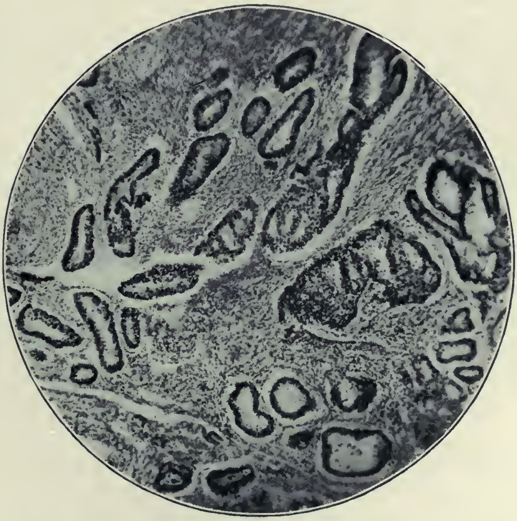


Section of an adenomatous growth of a simple nature which involved the cervix uteri. Large spaces are seen lined by a regular columnar epithelium; the spaces themselves contain mucin which has contracted during the processes of hardening. The spaces are probably derived from the glands of the cervix.

(From Specimen 2960 A. Museum, St. Bartholomew's Hospital.
Microphotograph by C. H. Cosens.)

Fig. 124.

ADENOMA MALIGNUM OF THE CERVIX.



Section of a growth which occupied the cervix uteri. The section shows, in places, groups and columns of cells more or less regularly arranged in acini and resembling the normal glands of the cervix, but they have involved the tissues of the cervix very deeply. In other areas, especially to the right and upper part of the section, the cells have undergone processes of proliferation identical with cancer. The growth is probably a malignant adenoma.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

like variety, are said to occur, but without microscopical examination a positive diagnosis between sarcoma and a squamous-celled carcinoma cannot be made.

Adenoma of the Cervix.—This is of the greatest interest pathologically. Our knowledge of the subject is small, and the views held at the present time are conflicting. Adenoma seems to be in some cases quite simple (fig 123. also figs 76. 77). The glandular erosion is an example of this (see chapter on Endometritis) ; mucous polypus, again, is an adenoma, and yet there are a few cases on record where actual glandular invasion [1] of the cervix has taken place, and although such invasion, even of the deeper structures, keeps to the type of adenoma, it is more than probable such tumours have malignant properties (fig. 124). Williams [2] describes a case in which the upper part of the vagina was occupied by a large mass which was the diseased cervix, the tumour being formed of innumerable glands, with a scanty framework between them of fibrous tissue. The glands were of every shape and form, lined with a single layer of columnar epithelium, and the cells presented no cancerous change.

Winckel [3] describes a condition of adeno-myxoma of the cervix. In St. Bartholomew's Hospital Museum [4] is a tumour of the cervix full of large glandular cavities lined with a single layer of columnar epithelium, the main mass of the growth being like a cervical fibroid. Much depends in such cases on the after history, and if not actually maligant they seem to be on the borderland of that condition.

Etiology, Frequency, and Occurrence of Cancer of the Cervix.—In spite of the immense amount of work done, and the enormous literature on the subject, we yet know little of the causes of malignant disease of the uterus. We know that women are more liable to cancer than men, and, of such cases in women, the uterus is affected in a very large proportion. Simpson's [5] and Spencer Wells' [6] tables on the subject may be consulted. No age above twenty-eight excludes it (Williams).

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[1] McCann, *Trans. Obstet. Soc. Lond.* vol. xl. 1898, p. 2.

[2] Sir J. Williams, *Cancer of the Uterus*, p. 42.

[3] Winckel, *Lehrb. der Frauenk.* 1888, p. 430.

[4] Specimen, St. Bartholomew's Hospital, No. 2960 B. See also 2960 A.

[5] Simpson, *Clin. Lect. Diseases of Women*, 1872.

[6] Sir Spencer Wells, *Morton Lect.* 1888.

Gusserow's tables[1] of 3471 cases are also of great interest, the majority being between the ages of forty and fifty, thus :—

CANCER OF THE UTERUS DEVELOPED AT THE AGE OF—

17	years=	1 case.	40–50	years=	1196 cases
19	„ =	1 „	50–60	„ =	856 „
20–30	„ =	114 „	60–70	„ =	340 „
30–40	„ =	770 „	Above 70	„ =	193 „

Schroeder found that in 19,666 women who died of cancer, 6548 were affected with cancer of the uterus.

Of course in such analyses all varieties of cancer are included, probably sarcoma as well, but cancer of the cervix must be looked upon as a disease quite distinct from cancer of the body, or from sarcoma, either of body or cervix. Cancer of the body is a comparatively rare disease found under strikingly different conditions from cancer of the cervix, and may be put down at present at about 2–3 per cent. of all cases of cancer of the uterus.[2]

As regards **race**, Cullen[3] thinks cancer equally common in the negro, while in all countries cancer seems more frequent among the poorer members of the community.

As regards **child-bearing** different views are held. In cancer of the cervix, Winckel,[4] quoted by Haultain, says that the large majority of women with uterine cancer are married; of Winckel's cases only 1·7 per cent. were unmarried, and two-thirds of these had given birth to one or more children.

With reference to this point, Haultain, in an analysis of 100 consecutive malignant cases, finds only one unmarried woman (æt. 52), and she had sarcoma; seventy women, of whose cases the record was kept, had borne 412 children, and the total number of abortions of the seventy was 68.[5]

The writer gathers from Williams' cases that he does not think that pregnancy, or the characters of the labours, have any influence in the development of cancer of the cervix, and that age at the time of marriage, and the duration of married life, have no effect either.

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[1] Clarence Webster, *Diseases of Women*, p. 505. See also Pichot's statistics, *Thèse de Paris*, 1888; and Cullen, *Cancer of the Uterus*, 1900, pp. 174–175, 334.

[2] Haultain, Allbutt and Playfair, p. 656. [NOTE.—In the opinion of the Author, cancer of the body occurs more frequently than is at present supposed.]

[3] Cullen, *op. cit.* p. 337.

[4] Winckel, *Diseases of Women.*

[5] See also Cullen, *op. cit.* p. 336.

Williams[1] also speaks strongly on a point relative to this question, **laceration of the cervix,** and says that 'lacerations play no part in the causation of cancer of the uterus'; also, that if irritation be the cause of cancer, the disease should be frequently met with in procident uterus, but this again is not the case.

Haultain,[2] on the other hand, speaking of the factors which modify nutrition in connection with cancer, says that irritation from lacerations of the cervix and chronic cervical catarrh appear to predispose to malignant disease, and that the coincidence of epithelioma and multiparity is too frequent to be explained as a mere chance.[3]

Mitchell Banks,[4] speaking of traumatism in relation to cancer, says he does not assert that cancers of traumatic origin are at all frequent, but he believes in the possibility, and indeed in the strong probability, of their occurrence. Acute traumatic malignancy[5] is a well-recognised disease, at all events in the relation between injury and sarcoma; and Mitchell Banks says that 'if injury will, so to speak, on the spot start into existence a swift-growing tumour of the connective tissue, why should it not at a longer interval give rise to the slow-growing tumour of glandular epithelium?' Bumm, Winckel,[6] Berns[7] and others maintain that gonorrhœa may favour the development of carcinoma.

Heredity seems to play a very doubtful part in cancer, and many side issues have to be taken into account. Mitchell Banks,[8] speaking on the subject of heredity in relation to cancer of the breast, says that 'while the want of history of a cancerous tendency inherited from her progenitors is no proof that the patient's disease is not cancer, the existence of it among several near relations is distinct evidence in the direction that it is so, though figures seem to show that the inherited infectivity of cancer is not strong.'

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[1] Sir J. Williams, *Cancer of the Uterus*, p. 81: 'Lacerations of Cervix in relation to Cancer.'

[2] Allbutt and Playfair, *op. cit.* p. 660: 'Ubi stimulus, ibi fluxus.'

[3] See also Cullen, *op. cit.* p. 651, 'Trauma as a Cause of Cancer.'

[4] *Lancet*, vol. i. 1900, p. 689.

[5] Sir W. M. Banks, *op. cit.* p. 689; Bowlby, *Surg. Pathol.* 4th ed. p. 159.

[6] Winckel, *Diseases of Women.*

[7] Berns, *Deutsche Zeit. für Chir.* vol. xxvii. p. 5.

[8] Sir W. M. Banks, *Lancet*, vol. i. 1900, p. 688.

Cullen,[1] however, states that heredity seems to play no part as a factor in the incidence of cancer of the uterus.[2]

Finally, with regard to etiology of cancer we are still in the dark. Cohnheim's theories of the embryonic origin of cancer have more or less been abandoned. He held that during fœtal life portions of the epithelium become nipped off and included in the connective tissue, these portions in later life, if stimulated to activity, giving rise to a cancerous growth.

Ribbert's[3] theory seems also untenable. The connective-tissue cells, he says, increase to such an extent that they invade the epithelial layer and nip off epithelial cells or groups of cells, such isolated epithelium being capable of producing cancer.

Possibly the views of Hausemann[4] and others that cancer is primarily a disease of the epithelium is correct, but we are totally ignorant of the cause of this cell alteration.

Experimental Inoculation of cancer cells in man and animals has been met with negative results as a rule,[5] though clinically curious cases appear to occur in peritoneal inoculation in papillomatous ovarian cysts, and Cullen[6] reports a case where in papillo-cystoma of both ovaries development of papillomatous growths occurred in the abdominal wall along the track of the trocar which was used in tapping the tumour.

Implantation of cancer cells[7] (or cancer parasites) into wounds of the vagina during vaginal hysterectomy for malignant disease seems sometimes to occur, and great care should be taken at operations to avoid this possible accident.

Cases of epithelioma of the vulva are sometimes seen where the growth on one labium appears to have infected the opposite side, with which it was in contact.

Spread of Cancer.[8] (Figs. 120, 125.)—The evidences of cancer

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[1] Cullen, *Cancer of the Uterus*, p. 651. See also Bowlby, *Surg. Pathol.* 4th ed. p. 158.

[2] See, however, 'Etiology of Cancer,' *Lancet*, vol. i. 1901, p. 1413, and Gaylord, 'Protozoon of Cancer,' *Amer. Jour. of Med. Sciences*, May 1901.

[3] Cullen, *op. cit.* p. 653.

[4] Cullen, *op. cit.* p. 657.

[5] See, however, Sir W. M. Banks, *Lancet*, vol. i. 1900, p. 991, on the results of Russell's inoculation experiments in cats and dogs.

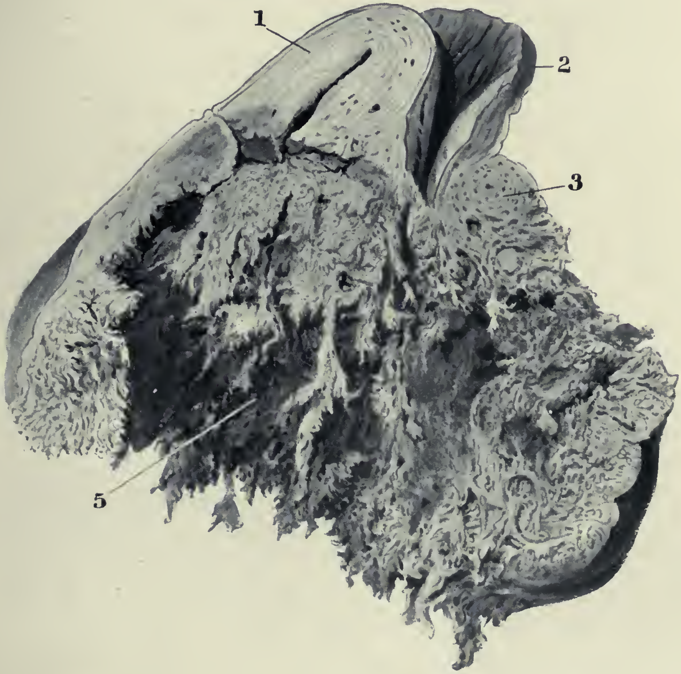
[6] *Op. cit.* p. 662; case 3323.

[7] Cullen, p. 663, quotes Hofmeier's and Leopold's cases. See also Banks, *op. cit.* p. 992.

[8] Cullen, *op. cit.* pp. 326–333; Lewers, 'After-Results in forty Cases of Vaginal Hysterectomy for Cancer of the Uterus,' *Lancet*, vol. i. 1901, p. 9.

Fig. 125.

VERY ADVANCED CANCER OF THE CERVIX.



A specimen showing the extension of malignant disease, beginning in the cervix. It has involved the bladder, which is extensively infiltrated, a large portion of its cavity being occupied by growth. Extensive sloughing has taken place.

1. The fundus uteri.
2. Wall of bladder.
3 Areas of cancerous growth extending into the bladder.
5. Mass of sloughing cancerous growth occupying the cervix and vagina. It also involved the rectum.

(Specimen 3011. Museum, St. Bartholomew's Hospital)

are hæmorrhage, more or less offensive discharge, and pain. The **hæmorrhage** comes from the cancerous surface; it is irregular and never a menorrhagia: menstruation [1] plays no part whatever in cancer; it is rarely profuse, unless some vessel be opened by ulceration. Hæmorrhage after the climacteric is extremely suggestive of cancer.

Discharge may or may not be offensive, it may be slight or profuse, and comes from the surface of the growth; and generally, if profuse, it means that a large cancerous surface exists, either as an ulcer or as a mass in the vagina.

Pain generally coincides with the invasion of the parametrium and in such cases cancerous parametritis may cause fixity of the uterus. Pain may be due to pressure on nerve trunks, and, if so, may extend down the legs. Ulceration in the vicinity of the internal os gives rise to the dull aching in the sacral region. Sometimes, in cases of cancer, the mass may block the internal os and **pyometra** [2] may result, the pain being hypogastric and spasmodic. Later still, there may be interference with bladder and bowel. If the peritoneum be involved, perimetritis may cause pain. Pain may be absent all along. If there be pain, it is generally worse at night, and is not relieved by lying down as is frequently the case with pain due to inflammatory conditions. Pains are more felt on the left side, and pain is a less infrequent early symptom than has hitherto been imagined.[3]

Rise of Temperature is met with in cancer either from septic infection, or inflammation of the peritoneum or cellular tissue.

Vomiting may occur early: if late in the disease it points to obstruction; but irritation of the bowel in cancer cases is sometimes seen apart from complete or partial obstruction, and may cause troublesome diarrhœa, the bowel being surrounded by a dense cellulitis.

Bladder troubles [4] (fig. 125) almost constantly occur, and mean that infiltration of the loose tissue between the bladder and the uterus has occurred, producing irritation and cystitis. Later, ulceration may occur with the formation of distressing fistulæ, or large masses of growth may protrude into the bladder.

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[1] Cullen, *Cancer of the Uterus*, 1900, p. 335.

[2] Cullen, *op. cit.*, 'Pyometra,' pp. 115–311. See also W. Tate, 'Three Cases of Pyometra complicating Cancer of Cervix,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 323; Zabloudovsky, *Sur les Rétentions Utérines*, Paris, 1900; chapter on 'Pyometra' in this work.

[3] Champneys, 'Pain in Pelvic Cancer,' *Trans. Obstet. Soc. Lond.* vol. xxii. 1880, p. 5.

[4] Bladder involved, two fifths of all cases of cancer; vagina involved, four fifths of all cases. Herman, *Diseases of Women*, p. 359.

If the **urethra**[1] be involved, retention of urine may result.

Cancerous infiltration of the parametric tissues may involve the **ureters**;[2] they are embedded, not displaced; if constricted, dilatation above the constriction results, and hydronephrosis, pyonephrosis, pyelitis and other evils ensue, producing obstructive suppression and uræmia. Haultain[3] points out the importance of the condition of the urinary organs in cancer cases, in regard to prognosis.

Later still, there may be recto-vaginal, recto-uterine, or uretero-vaginal as well as vesical **fistulae**, and the vagina becomes a mere cloaca, with terrible suffering, the patient becoming anæmic, uræmic, sapræmic, and wasted.

If large cancerous masses occupy the pelvis even **blood vessels** such as the iliac veins are pressed upon, producing œdema. There may be fulness of the veins over the front of the abdomen, ascites, thrombosis and phlebitis. The uterine veins may inflame, and pyæmia may result in this way.

Fatty degeneration of the heart may cause sudden death; lardaceous and fatty livers are sometimes found.

Causes of Death in Cancer.[4]—Hæmorrhage itself rarely kills: patients generally die from one or other of the complications of cancer, such as peritonitis, local or general, dysenteric ulceration or inflammation of the bowel, cystitis, pyelitis and uræmia, pulmonary embolism, pyæmia, bedsores, pneumonia and pleurisy from secondary growths, sudden failure of the heart, or gangrene of the lung from putrid emboli. The duration of life in uterine cancer is rarely more than 18 months.

Involvement of Lymphatic Glands, and Metastatic Deposits in Cancer of the Cervix.[5]—The lymphatic vessels of the vulva and lower third of the vagina go to inguinal glands; those from the middle third of the vagina and cervix to the iliac glands at the bifurcation of the common iliac arteries; those of the body

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[1] Herman, *Diseases of Women*, p. 363.

[2] Lanceraux, *Ann. des Mal. des Org. Gén.-urin.* 1884.

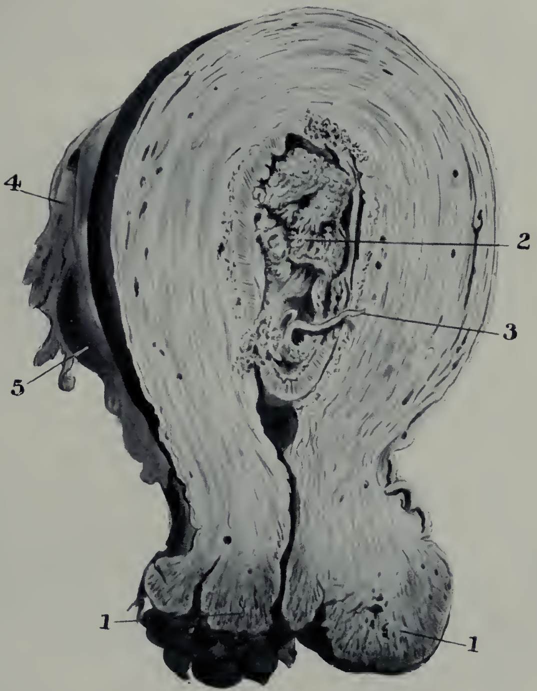
[3] Haultain, Allbutt and Playfair, *op. cit.* p. 667.

[4] Herman, *op. cit.*, p. 366.

[5] Cullen, *op. cit.* p. 328; Sir J. Williams, *Cancer of the Uterus* (1888), case xvi.

Fig. 126.

EARLY PREGNANCY AND CANCER OF THE CERVIX.



A uterus removed by vaginal hysterectomy for cancer of the cervix. The external os is patulous and hypertrophied, and is the seat of a cancerous growth. The whole uterus is enlarged, and shows the changes due to pregnancy towards the end of the first month.

1, 1. The cervix enlarged by cancerous infiltration. The growth is a carcinoma. The surface towards the vagina is ulcerated.
2. The membranes and decidua of a pregnancy *in situ*, at about the end of the first month.
3. Umbilical cord.
4. Fallopian tube.
5. Ovary.

(Specimen 3006 D. Museum, St. Bartholomew's Hospital.)

and fundus pass along the broad ligament with the ovarian vessels to the lumbar glands below the kidney. Lymphatics also run in the round ligament from the fundus to the inguinal gland. The obturator gland has no connection with the uterus as is sometimes stated.

In cancer, glands are attacked late, and only when fixity of the uterus occurs, and in about one third of the cases. (Herman.) Recurrence after removal of cancer occurs, not in glands, but locally in cellular tissue, the cicatrix, bladder, vagina, or peritoneum.

Metastatic deposits[1] are rarer still in cancer of the uterus, if they occur at all (less than 10 per cent., Herman). The liver, lungs and kidneys are mostly affected.

Winter[2] found in 202 cases of recurrence only 9 real examples of metastasis, and only 2·5 per cent. of all women operated upon had metastasis at all. The stomach, lungs and ovaries were the sites affected.

Local infection seems to be proved in some cases by prolonged contact of cancerous masses with other tissues, also by infection of the adjacent peritoneum possibly at the time of operation.

Cancer of the Cervix and Pregnancy.[3] (Fig. 126.)—This is uncommon, but in some cases may lead to dangerous obstruction to labour. Cancer of the cervix, if not advanced, may be of little, if any, hindrance to conception. Cancer of the cervix proper (the glandular carcinoma) is most marked in its effects, owing to the early invasion of cellular tissue. Epithelioma spreads rather to the vagina, and does not necessarily affect dilatation of the cervix.

The stimulus of pregnancy largely adds to the rapidity of the growth of cancer, but after delivery the growth may be retarded for a time.[4] The cancer in some degree shares the softening of the cervix which results from pregnancy. Probably cancer causes intra-uterine death of the ovum, or its premature expulsion, in a fair proportion of cases.

[1] Delbet, *Traité de Chirugie*, 2nd ed. vol. viii. p. 195; Cullen, *op. cit.* p. 331.

[2] Winter, 'Ueber die Recidive des Uterus Krebses,' *Verhandl. der Deutschen Gesellschaft für Gynäk.* 1893.

[3] Cullen, *Cancer of the Uterus*, p. 611, and literature: Cohnstein, *Arch. für Gynäk.* 1873, Bd. V. p. 366; Pinard, *Clinique Obstét.* 1899, p. 119; Delbet, *op. cit.* p. 215; Galabin, *Diseases of Women*, 5th ed. 1900, p. 468.

[4] Dakin, *Handbook of Midwifery*, p. 290.

At full time, cancer may be incipient or advanced; if the former, and the cancer be soft, dilatation of the cervix often takes place as usual; if advanced, the cervix may be hopelessly involved, dilatation is impossible and labour is obstructed. Besides the cervix, there may be severe infiltration of the cellular tissue and immovable masses of growth in the pelvis, or large masses of cancer may project into the vagina. Sometimes no trace of anything like the cervix can be made out.

Cancer of the body of the uterus, apart from Deciduoma (so-called), is a very rare occurrence with pregnancy, as the disease generally occurs at a time of life when age precludes it, and sterility for that reason is the rule.

CANCER OF THE BODY OF THE UTERUS.

This includes, besides the ordinary cancer of the body, Sarcoma, with its sub-class Deciduoma, and Adenoma malignum.

Primary Carcinoma of the Body.—This is certainly a much rarer disease than cancer of the cervix, but it probably occurs more frequently than is supposed.

It develops in much older women than cancer of the cervix: generally patients have passed the menopause, and sterility, as stated above, is thus a factor in the history.

Carcinoma of the body of the uterus[1] appears to occur in two more or less different forms:

(*a*) Originating from the glands of the mucous membrane, the **diffuse** form of cancer.

(*b*) Originating more deeply in the uterine tissues and forming a **localised** mass of growth.

The diffuse form is said to be the commoner: most books describe the local or circumscribed form, but as originating in the deep uterine tissues it is very rare, and such cases are more probably sarcoma (*q. v.*); Williams[2] says he has not met with an instance of the circumscribed form.

The **diffuse form** originates in the mucosa, either from its glands or the superficial epithelium. Starting in the glands, it gradually becomes superficial towards the uterine cavity, and

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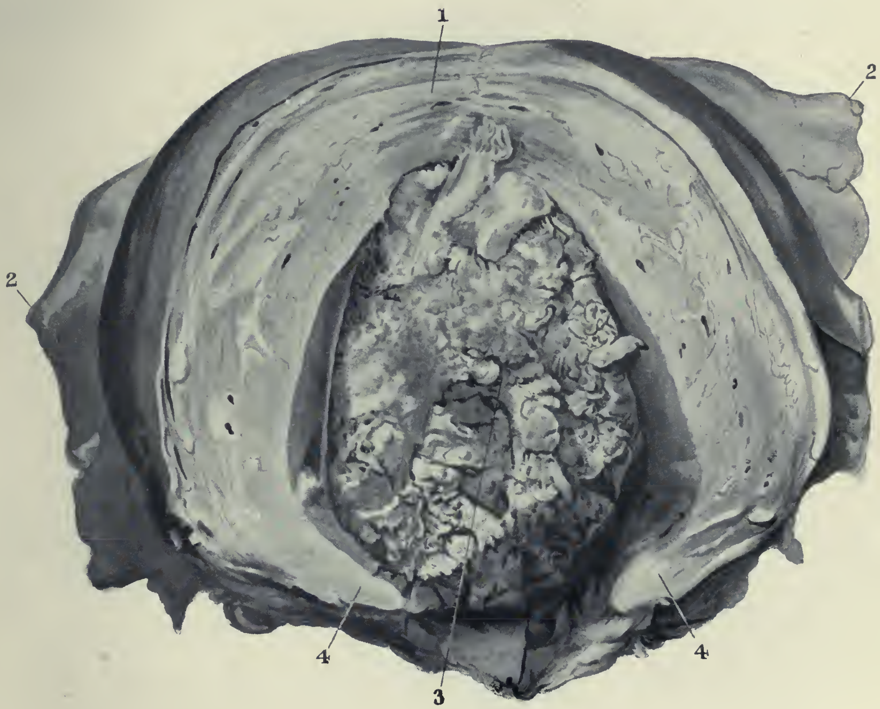
[1] Cullen, *Cancer of the Uterus*, 1900, p. 353. See also Handfield Jones, *Brit. Med. Journ.* vol. i. 1901, p. 136.

[2] Sir J. Williams, *Cancer of the Uterus*, p. 97.

FIG. 127.

CANCER OF THE BODY OF THE UTERUS.



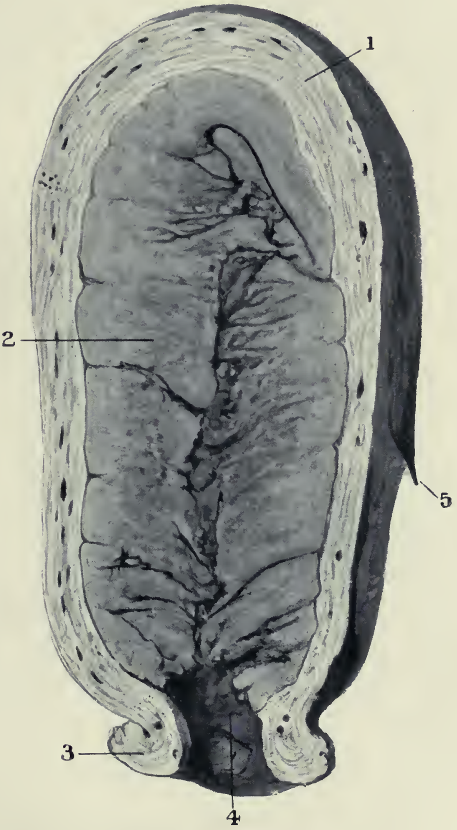
The upper part of a uterus removed by abdominal hysterectomy; it has been laid open to show a fungating growth filling the cavity of the body. Microscopically it is an adenoid carcinoma.

1. Body of uterus laid open to show the growth on its anterior wall.
2, 2. Fallopian tubes.
3. Mass of fungating growth more or less localised. Some parts of its surface are necrotic and very friable.
4, 4. Region of the internal os. The cervix was not involved.

(Specimen 3011 A. Museum, St. Bartholomew's Hospital, Section xliii. (3011 A), Pathol. Series.)

FIG. 128.

CARCINOMA OF THE BODY OF THE UTERUS.



A uterus the body of which is uniformly enlarged. The cavity is everywhere occupied by a mass of cancerous growth, which involves the walls. The growth is breaking down on its free surface. The canal of the cervix is dilated.

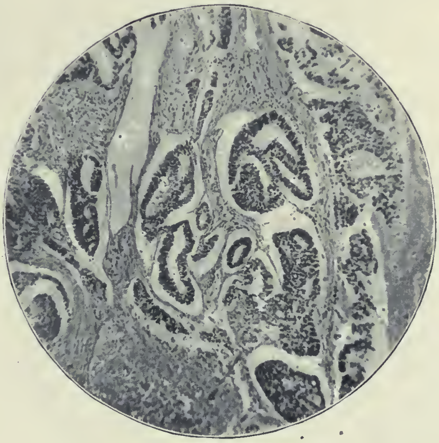
1. Muscular wall of uterus.	4. External os, much dilated.
2. Mass of cancer filling the cavity.	
3. Cervix.	5. Limit of peritoneum anteriorly.

Note.—In the drawing too much distinction has been made between the growth and the muscular wall of the uterus.

(Specimen 3010 B. Museum, St. Bartholomew's Hospital.)

Fig. 129.

COLUMNAR-CELLED CARCINOMA OF THE BODY OF THE UTERUS.

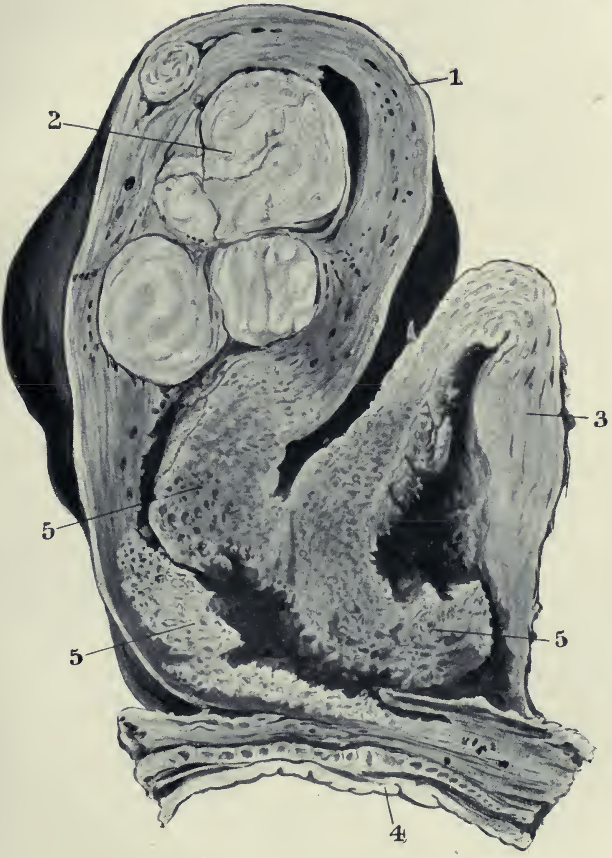


Section of a cancerous growth which sprang from the body of the uterus. Large masses of atypical columnar epithelium are seen involving the deeper layers of the uterine wall; they are enclosed in irregular spaces, the cells of which in some places have dropped out during the processes of hardening and cutting. In some areas, on the right of the section, columns of cells have penetrated to the muscular layers.

(Microphotograph by C. H. Cosens.)

Fig. 130.

FIBROIDS AND CANCER



A uterus showing three distinct fibroids in its upper portion. Below, there is extensive malignant disease of the cervix, which has involved the vesico-vaginal septum and posterior vaginal wall.

1. Fundus uteri.
2. Mass of ordinary uterine fibroids.
3. Bladder.
4. Rectum.

5, 5, 5. Extensive area of malignant disease of the cervix, involving bladder and vagina.

(Specimen 3004 a. Museum, St. Bartholomew's Hospital.)

at the same time penetrates deeper and deeper into the uterine wall; it may sometimes assume a polypoid form[1] (fig. 127), as described in the chapter on 'Endometritis'; this may break down, leaving a mass of cancer growing from the uterine wall.

Generally the whole of the inner surface of the body is affected (fig. 128) or only a small part escapes. Whether this is the case from the beginning or as the result of the mode of growth is not known; the surface is villous, rough and ulcerated, or covered with warty nodules, sessile masses, or polypi. (Williams.) The cervix is rarely affected, and then very late. Sometimes the uterus is markedly enlarged, and the muscular walls thick, or the whole uterus is invaded even to the peritoneal surface, where bulging nodules occur. The cavity of the uterus may be only slightly enlarged, or the finger may pass into a big necrotic cavity.[2]

In this glandular type of carcinoma sections show the uterine glands everywhere blocked and filled with proliferation of their epithelial elements, especially in the deeper parts. The cancer in a way imitates uterine glands, later the infiltration and glandular proliferation is more extensive and 'lawless'[3] and masses of cells are found invading the stroma everywhere (fig. 129) both towards the surface and deep in the muscle, even penetrating to the peritoneal surface or spreading along the Fallopian tube.

If a fibroid occur in such a uterus it may be invaded and destroyed by cancer cells, but this is not malignant degeneration of a fibroid,[4] as fibroids and cancer may co-exist. (Fig. 130.

Another form of cancer of the body assumes a superficial papillary type. Cullen[5] states that there is a distinct and progressively increasing proliferation of the surface epithelium, beginning with small outgrowths consisting of a few cells, next a larger outgrowth containing stroma which shows marked branching, and finally an excessive proliferation of the epithelium with the formation of many new glands.

The papillary form of cancer of the body is very similar to epithelioma of the cervix, and many cases have been reported lately in which tumours of this nature[1] have been observed; to all intents and purposes they are epitheliomata; and it is even possible that both forms may occur in the same case and be but stages or different developments of the same disease. Pfannenstiel[2] recognises the two types of cancer of the body as an adeno-carcinoma, and adeno-carcinoma papillare (papillary epithelioma): the latter taking the form of superficial proliferation with necrosis and ulceration, forming a tumour comparatively late in its development.

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[1] Sir J. Williams, *op. cit.* p. 97; Cullen, *op. cit.* p. 354.

[2] It is not at all unusual for cancer of the body of the uterus to undergo necrosis, and a putrid discharge results.—AUTHOR.

[3] Cullen, *op. cit.* p. 359.

[4] Sir J. Williams, *op. cit.*; Cullen, *op. cit.* pp. 410, 439, and case 31.

[5] Cullen, *op. cit.* p. 356.

Papillary growths of the body may be of a simple nature, but they may also assume a malignant type[3] and invade the uterine wall, showing the tendency which simple adenomata[4] and papillomata have to become malignant when affecting the uterus.[5]

In cancer of the body hæmorrhage is the constant accompanying factor; it may be very profuse or slight. Offensive discharge is often present. Pain is a common symptom and may occur very early. In those cases where the uterus contains a large mass of cancer, painful uterine contraction may be set up as the uterus endeavours to expel the growth. There is never the pain like cancer of the cervix where the cellular tissues are involved. In late stages of cancer of the body peritonitis may account for the pain, as in a case seen by the writer, in which perforation of the uterus had taken place and a large fœtid pelvic abscess[6] formed, shut in by peritoneal adhesions, which emptied itself from time to time into the uterus. Pyometra[7] occurs with cancer of the body as well as in cancer of the cervix, and may also cause much pain.

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[1] Von Rosthorn, quoted by Cullen, p. 471; Zeller, *Zeit. für Geb. und Gynäk.* 1884–85, vol. xi. p. 56. Delbet, *Traité de Chirurgie*, 2nd ed. vol. viii. p. 199, quotes two cases of pavement epithelioma of the body, and suggests that they may arise from squamous epithelium of the body, into which the columnar epithelium is sometimes changed.

[2] Sinclair, *op. cit.*, Allbutt and Playfair, p. 714.

[3] Routh, 'Malignant Papilloma of Uterus,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 5; Targett's report, *ibid.*

[4] Eden, 'Specimen of Adenoma of the Uterus,' *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 2.

[5] Roberts, *Trans. Obstet. Soc. Lond.* vol. xlii. 1900, p. 267; *id. Lancet*, 1900, vol. ii. p. 1075.

[6] See also Sir J. Williams, *Cancer of the Uterus*, case 28; Cullen, *op. cit.* p. 413.

[7] Cullen, *op. cit.* p. 409; Mdlle. Cécile Zabloudovsky, *Sur les Rétentions Utérines*, Paris, 1900.

In spite of even great involvement of body of the uterus, fixity is very uncommon, and the case may run on for a very long time without lymphatic infection or metastasis, the uterus remaining mobile; the tubes and ovaries are rarely affected; bowel *may* be adherent to such a uterus and this has to be thought of in removal of the uterus. The lumbar glands situated on a level with the lower border of the kidney, in front and on either side of the vena cava and aorta, may be affected; the inguinal glands may be involved if the cancer affects the uterine horns, their lymphatics passing down the round ligament.[1] The prognosis in cancer of the body, both as regards the disease and the results of operation, is much more favourable than in cancer of the cervix.

SARCOMA AND DECIDUOMA

Sarcoma occurs in the uterus (figs. 131, 132) probably more commonly than is thought, and many cases that used to be described as cancer were probably of this nature. Much light has been thrown upon this subject of late, especially in regard to 'Deciduoma' as first described by Sänger in 1883,[2] of which more will be said later.

Apart from Deciduoma and its connection with pregnancy or abortion, Sarcoma of the uterus may also occur, as either

(*a*) **Circumscribed.**
(*b*) **Diffuse.**

Great confusion exists in the descriptions of sarcoma in most books, and the subject is exceedingly difficult and complex [3]

The **circumscribed** form of sarcoma [4] is the commoner, and bears a striking resemblance to the growth of uterine fibroids: in fact most of the cases observed have been diagnosed as such, at all events in elderly patients. In younger women the rapid increase in the size of the uterus, with profuse hæmorrhage and discharge should lead to a suspicion of the condition, and examination of particles removed by a curette carefully made. Ultimately the whole uterus becomes fixed, secondary tumours rapidly form in the pelvis, and death occurs from metastatic deposits in the viscera, especially the lungs. The disease starts from the connective tissue of the uterine wall, or from the muscular elements. The sarcoma may be a round, mixed-celled, or fibro-sarcoma. Such cases are frequently accompanied by profuse discharge, and by the passage of necrotic, œdematous, or sloughing, portions of the growth; if portions are removed by the curette the disease rapidly recurs (fig. 131). Sarcoma is often extremely vascular, but the blood vessels are of the embryonic type, there is generally no capsule to such a growth, and the whole thickness of the uterine wall may be invaded.

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[1] See Poirier, *Lymphatiques des Organes Génitaux de la Femme*, Paris, 1890; and Cullen, *op. cit.* p. 421.

[2] Sänger, *Centralb. für Gyn.* 1889 and 1894, vol. xliv., and *Verhandl. der Deutschen Gesellschaft für Gynäk.*, 1892.

[3] 'Il est impossible pour le moment de donner une description du Sarcome de l'utérus,' Delbet, *Traité de Chirurgie*, 2nd ed., tome viii. p. 186.

[4] Whitridge Williams, 'Contributions to the Histology and Histogenesis of Sarcoma of the Uterus,' *Amer. Journ. Obstet.* 1894, vol. xxix. p. 721; and Cullen, *Cancer of the Uterus*, 1900, p. 547.

The greatest difficulty arises in the question of cases of sarcoma which are supposed to be due to malignant degeneration of uterine fibroids (fig. 132). See chapter on Uterine Fibroids.

The great point is, Can such a transformation take place?[1] or is not the tumour from the first a sarcoma? The subject is shrouded with most perplexing obscurity; the writer is of the opinion that probably such tumours are sarcomata from the beginning. Reported cases are inconclusive. Because a woman has a tumour for even many years, which is diagnosed as a fibroid, and then, suddenly perhaps, late in life, such a tumour grows and takes on malignant changes, and death ensues with secondary sarcomatous deposits in the viscera corresponding to the uterine growth, it does not prove anything more than that the growth is a sarcoma, or that the growth originally diagnosed as a fibroid might not have been a sarcoma from the first. Then, again, the microscopical investigation of fibromyoma and fibro-sarcoma is ever a subject of discussion and doubt, and their exact differences remain difficult to prove.

Sinclair, in Allbutt and Playfair's' 'System of Gynæcology,'[2] thinks that fibro-sarcoma (which is the commonest form of sarcoma of the uterus) is due to the transformation of fibro-myoma into sarcoma, and that the so-called œdematous fibroids whose growth is not influenced by oöphorectomy are of this nature.

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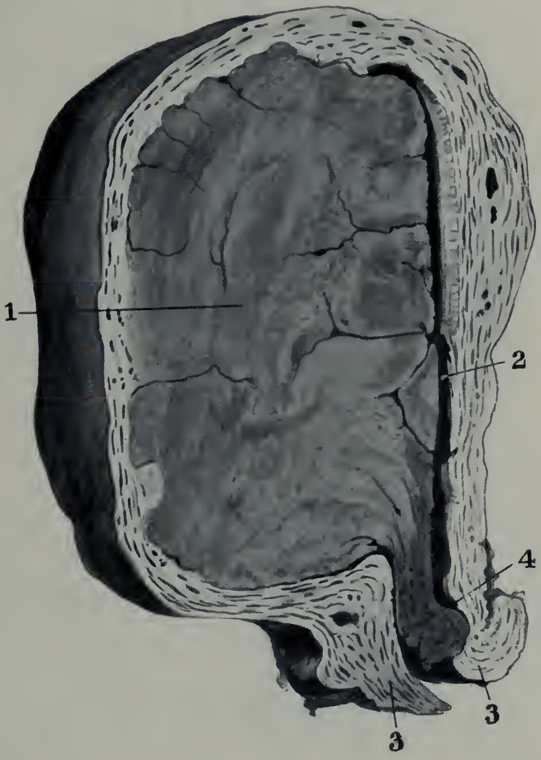
[1] P. Delbet, *op. cit.* p. 186, believes that fibro-sarcoma is a transformed fibromyoma.

[2] P. 723.

Fig. 131.

SARCOMA OF THE BODY OF THE UTERUS.



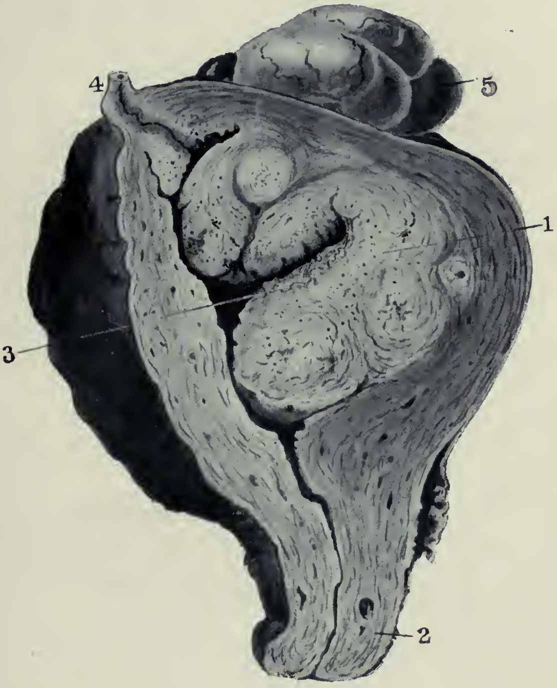
A uterus removed by vaginal hysterectomy, showing the body almost everywhere involved by a mass of soft growth which fills the cavity. A portion of this occupies the cervical canal. The walls of the uterus are invaded nearly to the peritoneal surface. The growth is a mixed-celled sarcoma, consisting of oval and spindle-shaped cells.

1. Mass of soft growth filling the uterus.
2. Cavity of uterus.
3, 3. Cervix.
4. Mass of necrotic growth hanging in the cervical canal.

(Specimen 3015 A. Museum, St. Bartholomew's Hospital. Griffith, 'Trans. Obstet. Soc. Lond.' vol. xli. 1899, pp. 233 and 371, Case 2.)

FIG. 132.

SARCOMA OF THE UTERUS (ASSOCIATED WITH SUBPERITONEAL FIBROIDS).



Specimen of a uterus the cavity of which is occupied by a sarcomatous growth. Growing from the fundus is a group of subperitoneal fibroids. The uterus was removed by vaginal hysterectomy, but the patient subsequently died with secondary growths in the lungs and thoracic walls. There is no proof that the growth arose from sarcomatous degeneration of a fibroid.

1. Sarcomatous growth in the fundus, invading the uterine wall.
2. Cervix.
3. Portion of tumour undergoing necrotic changes.
4. Fallopian tube.
5. Mass of subperitoneal fibroids.

(Specimen 3015 A.2. Museum, St. Bartholomew's Hospital. Griffith, 'Trans. Obstet. Soc. Lond.' vol. xli. 1899, pp. 232 and 371, Case 1.)

Whitridge Williams[1] holds that fibro-myomata may be transformed in sarcoma. Dührssen, quoted by Sinclair (p. 724), also reports a case of this sort.

Champneys[2] says he has never seen a case in which a well marked fibroid has taken on a malignant growth.

The writer has seen and investigated several supposed cases,[3] but has come to the conclusion that the subject is not proved as yet; and that when fibroids take on rapid growth after years of quietude, such growth is usually caused by cystic or œdematous degenerative changes (fig. 71), or the fibroid has become suddenly infected and inflamed, and that when undoubted sarcoma occurs there is no proof that the tumour was not a sarcoma from the first.

Large masses of sarcoma growing from the fundus may produce inversion. Targett has reported a case occurring in a single woman of 79.[4]

Diffuse Sarcoma[5] of the endometrium is described, and resembles exactly carcinoma of the body in its symptoms, the microscope only revealing the difference. Some growths are said to be in an intermediate condition[6] between carcinoma and sarcoma (?), secondary growths in viscera pointing to sarcoma.

Deciduoma Malignum. (Figs. 133, 134.)—This was first described by Sänger[7] of Leipsig. Pfeiffer independently in 1893 described the same condition. It is a variety of uterine sarcoma, which in its microscopical characters so strongly resembles decidual tissue that it has become the rule to speak of this condition as 'deciduoma.' The disease[8] is liable to occur in the endometrium within a few weeks or months of abortion or labour; the chief characters of the condition being oft-recurring, profuse hæmorrhage, great emaciation, enlargement of the uterus, and the appearance of secondary nodules in the thoracic and abdominal viscera; the disease taking a rapid and fatal course.[1]

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[1] *Amer. Journ. Obstetrics*, vol. xxix.

[2] *Lancet*, January 20, 1900, p. 150.

[3] For other cases see Routh, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 367 (see fig. 71); Griffith, *ibid.* 1899, pp. 232–371, and plates v. and vi. (see figs. 131, 132)

[4] Targett, *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 285.

[5] Delbet, *op. cit.* vol. viii. 2nd ed. p. 185, 'Sarcome de la Muqueuse.'

[6] Sinclair, *op. cit.* Allbutt and Playfair, p. 723.

[7] Sänger, *op. cit.*; Mayer, *Virchow's Archiv*, vol. lxvii.; and Cullen, *Cancer of the Uterus*, 1900, p. 595.

[8] Bland-Sutton and Giles, *Diseases of Women*, p. 203.

Sänger's[2] latest papers review the whole subject. Gottschalk[3] and Marchand[4] have also written extensively on the subject. Sinclair (Allbutt and Playfair, 'System of Gynæcology') gives a summary of Marchand's views in the article on Malignant Disease of the Uterus (p. 736). Marchand considers the growths to be rather epithelial than connective tissue tumours, and proposes the name 'serotinal tumour' for the word 'deciduoma' (or sarcoma deciduo-cellulare), which Sänger gives to the disease.

In England[5] many cases of the same kind have also been reported, and the discussion of the subject, with cases, before the Obstetrical Society of London in 1896 is worthy of the closest attention. Malcolm, Rutherford Morrison,[6] Herbert Spencer,[7] and others,[8] reported cases.

Most of the cases resembled each other. Within a few weeks after abortion or labour at term, hæmorrhage and discharge came on, a growth rapidly appeared in the uterus, with secondary growths in the viscera. The ages[9] of the three cases were between 27 and 35 years, death occurring in each case very rapidly after the growth appeared.

In the report of Malcolm's case the uterus was enlarged and fixed, and there were deposits in the pelvis, lungs and mediastinal glands. The growth was a sarcoma, the cells varied in size and shape, most being large, with a single nucleus; besides these, there were a few large irregular shaped masses of protoplasm, with several or many nuclei. These cell areas existed mostly in the periphery of the nodules, the centre and greater part being composed of blood corpuscles, entangled in a fibrous

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[1] For 'Metastases in Deciduoma' see Dorland's table, quoted by Cullen, and *Cancer of the Uterus*, p. 597.

[2] Sänger, 'Ueber Sarcoma Uteri deciduo-cellulare,' *Centralb. für Gyn.* 1889; *Archiv für Gyn.* 1894, vol. xiv.

[3] Gottschalk, 'Deciduoma Malignum,' *Berlin Klin. Woch.* 1893.

[4] Marchand, *Monat. für Geb. und Gynäk.* 1895, vol. i.

[5] Malcolm, 'Note on a Case of Malignant Disease of the Uterus with numerous Deposits in the Lungs, and Death, following an Abortion,' *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 125.

[6] Rutherford Morrison, 'A Case of Deciduoma Malignum occurring in England,' *ibid.* p. 130.

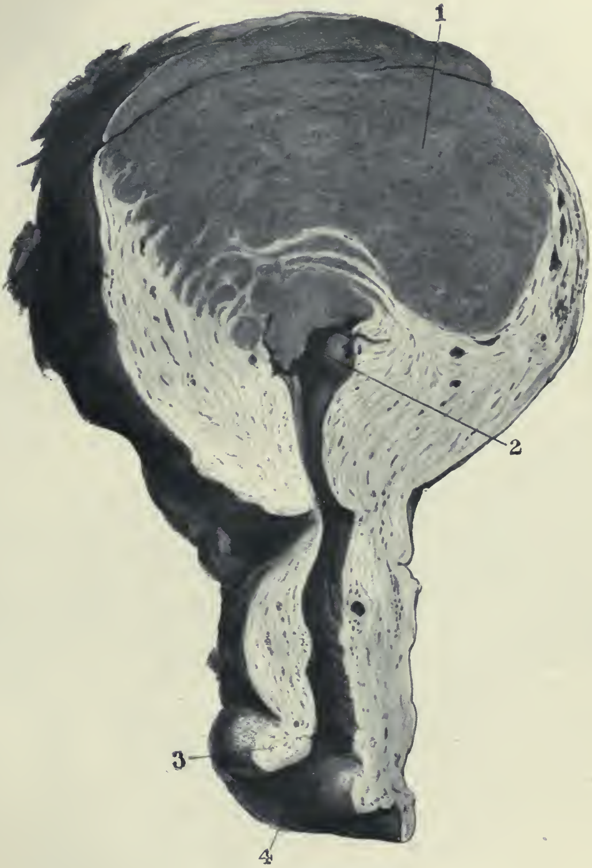
[7] Herbert Spencer, 'A Case of Deciduoma Malignum,' *ibid.* p. 135.

[8] Eden, 'Deciduoma Malignum: a Criticism,' *ibid.* p. 149; discussion, p. 171.

[9] See also Cullen, *op. cit.* p. 598.

FIG. 133.

SARCOMA OF THE BODY OF THE UTERUS (DECIDUOMA MALIGNUM?).



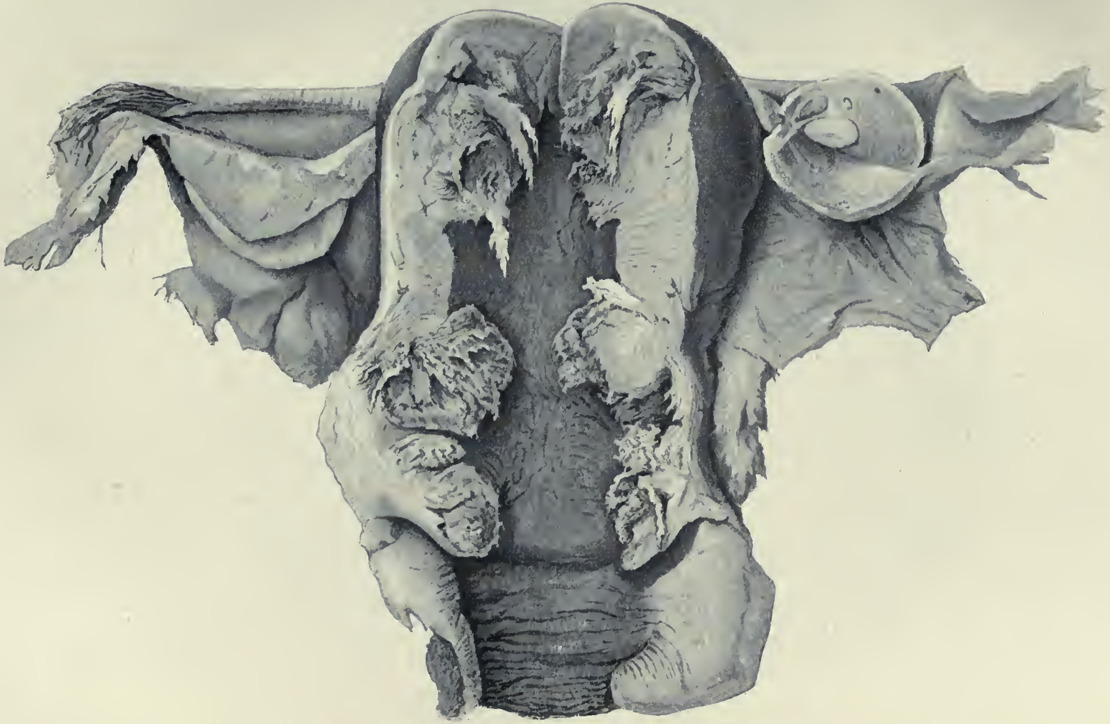
A uterus, the upper part of which is much enlarged by (when fresh) a red vascular softened diffused growth. The patient was delivered of a child seven months before her death. She died suddenly with dyspnœa and hæmoptysis. There had been severe uterine hæmorrhage for some weeks. There were similar growths in the lungs, lumbar glands, and anterior wall of the vagina. The growth is a sarcoma, with much extravasation of blood.

1. Mass of dark-coloured sarcomatous growth.
2. Portion which is ulcerating into the cavity of the uterus.
3. Cervix.
4. External os uteri.

(Specimen 3015 A. Museum, St. Bartholomew's Hospital.)

Fig. 134.

SARCOMA OF THE UTERUS (DECIDUOMA MALIGNUM).

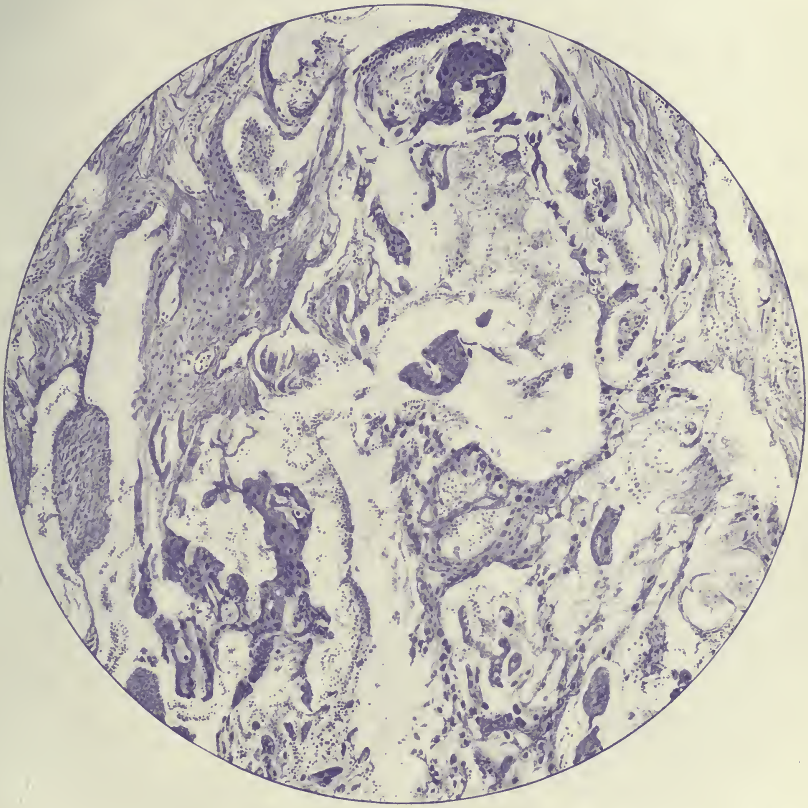


A uterus opened posteriorly, showing a malignant growth in the body and the cervix. The lower part of the body and the anterior wall of the cervix are not affected by the growth. There were secondary growths in the lungs, a section of which is shown in fig. 138.

(Herbert Spencer, 'Trans. Obstet. Soc. Lond.' vol. xxxviii. 1896, p. 144.)

FIG. 135.

DECIDUOMA MALIGNUM. (LOW POWER, 1-INCH OBJ.)



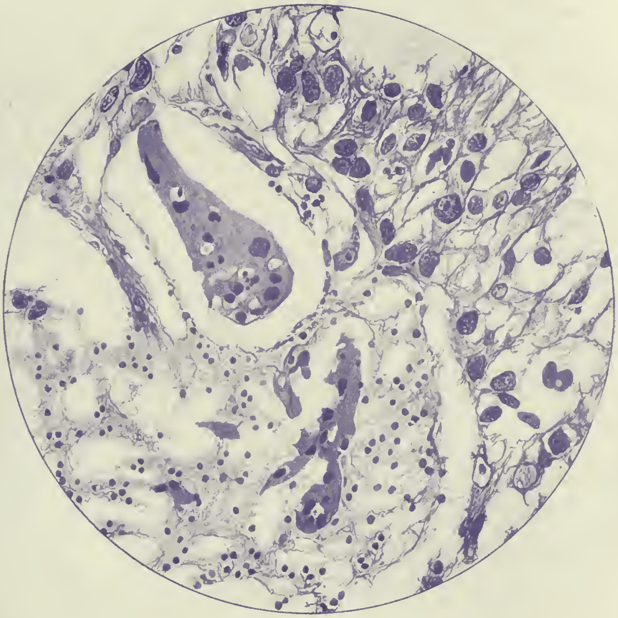
A microscopic section of the growth in the body of the uterus in fig. 134, showing a piece of the tumour penetrating, in the shape of a wedge, into the muscular wall, which is seen on each side enclosing the apex of the wedge.

Extensive masses of syncytium are seen, and (on the left) the abrupt line of demarcation between the syncytium and the uterine tissue is shown.

(Herbert Spencer, 'Trans. Obstet. Soc. Lond.' vol. xxxviii. 1896, p. 148, plate ii.)

Fig. 136.

DECIDUOMA MALIGNUM. (High Power, $\frac{1}{6}$-inch obj.)

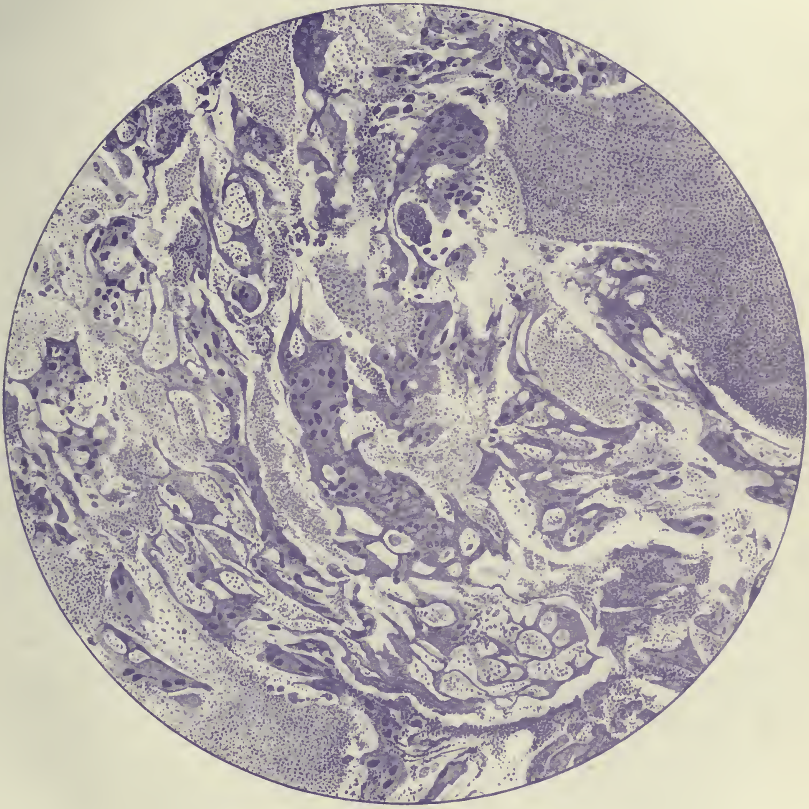


A microscopic section of the growth in the body of the uterus, from case in fig. 134, showing syncytium, individual cells, and intercellular reticulum.

(Herbert Spencer, 'Trans. Obstet. Soc. Lond.' vol. xxxviii. 1896, p. 148, plate iii.)

Fig. 137.

DECIDUOMA MALIGNUM. (Low Power, 1-inch obj.)

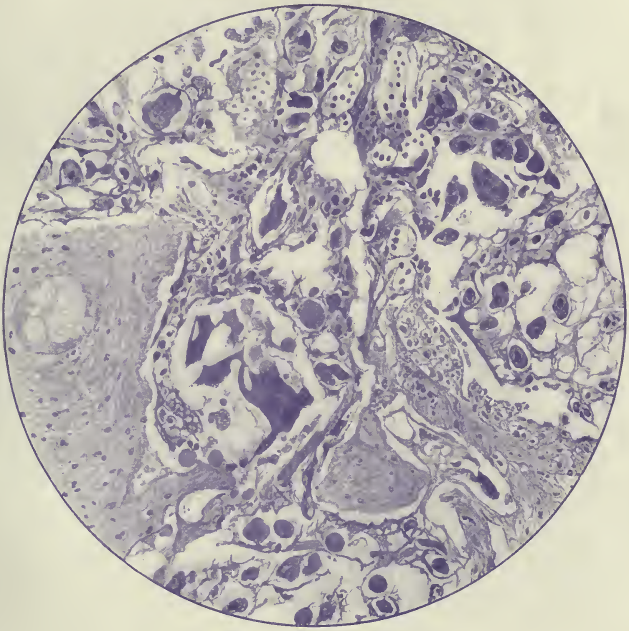


A microscopic section of the growth in the cervix of the uterus, in the same case as figs. 134, 135, 136, showing extensive masses of syncytium (in places vacuolated and filled with blood); also infiltration of leucocytes, of which a large collection is seen at the upper part of the section.

(Herbert Spencer, 'Trans. Obstet. Soc. Lond.' vol. xxxviii. 1896, p. 148, plate iv.)

Fig. 138.

DECIDUOMA MALIGNUM. (High Power. $\frac{1}{6}$-inch obj.)



A microscopic section of the growth in the lung, from the same case as figs. 134–137, showing intra-alveolar growth. Several small masses of syncytium are seen.

(Herbert Spencer, 'Trans. Obstet. Soc. Lond.' vol. xxxviii. 1896, p. 148, plate v.)

coagulum. The growths in the lung were quite similar; in some places there were vacuoles like thin-walled vessels. The uterus was 4½ inches long, and 3 inches across the fundus, there was a mass of new growth in the right side of the pelvis, in which were embedded the right ovary and ureter.

In Rutherford Morrison's case serious hæmorrhage began six weeks after a difficult labour at term; two months after the confinement a mass of growth was found in the uterus; the uterus was removed, but death eventually happened some months later from sarcoma of the lung. The uterus showed a growth the size of a tangerine orange infiltrating the upper and posterior walls, from which a large nodule projected on the peritoneal aspect. The Clinical Research Society, examining pieces of the growth early in the case, reported it as epithelioma.

In Herbert Spencer's case[1] (figs. 134, 135, 136, 137, 138) the patient was 27; she had had 2 children, the last 7 years ago; then a normal labour at term; 28 days afterwards masses of growth were passed, followed by repeated discharges of growth and blood; the disease ran a septic course, and terminated fatally within 10½ weeks of delivery.

At the necropsy an ulcerated and gangrenous growth was found at the placental site, the ulceration having nearly perforated the fundus; (fig. 134); there were secondary growths in the cervix and lungs.

The disease under the microscope was a large-celled sarcoma, the growth was of loose structure and made up of isolated multinucleated masses of large cells (the so-called syncytium), and of a loose tissue between the syncytial masses containing individual cells similar to those of the syncytium, which was vacuolated and contained hæmorrhages; the growth involved the muscular wall. The mass of the growth was sarcomatous, but the occurrence of the vacuolated nucleated masses of protoplasm (syncytium) amid the sarcomatous cells was remarkable. (Figs. 135, 136, 137.) In the lungs the growth was similar (*i.e.* sarcoma), but the syncytial masses were less frequent. (Fig. 138.)

Eden,[2] in criticising the subject of deciduoma malignum, pointed out that large cells like decidual cells are found in the uterus under various conditions, and do not prove the origin of the growth in the decidua. Sänger believes that such new growths arise from decidual cells, and Gottschalk that they arise from sarcomatous transformation of the stroma of retained chorionic villi. Marchand and Whitridge Williams [1] consider that the plasmodial masses are derived from the fœtal ectoderm of the villus (*i.e.* syncytial in origin). Mayer [2] reports a case in which there seems to be proof of the origin of a malignant growth in the retained myxomatous villi of hydatid mole (Eden).

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[1] Herbert Spencer, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 144. [The Author is much indebted to Dr. Spencer in being allowed to reproduce the drawing and beautiful microscopic sections of this case.]

[2] *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 149. Also see *Journ. of Path. and Bacter.* January 1896.

English authorities, however, believe that there is no evidence whatever that a retained placenta ever continues to grow after the death of the fœtus, and it is more difficult to believe that retained normal villi ever give rise to malignant growths, but relics of a vesicular mole might possibly do so.[3]

Sänger makes a great point of decidual cells as the originating point of deciduoma, but decidual cells (fig. 139) have no specific characters whatever; [4] they are modified connective tissue corpuscles, and cells exactly like them are found in many connective-tissue tumours in other parts of the body, and in the muscular wall of the puerperal uterus of the rabbit [5] and other animals during normal involution. The decidual 'cell' is not pathognomonic of pregnancy, but the decidua is.

The next important question is whether the plasmodial masses observed are necessarily from the fœtal elements of a villus, as is maintained (*i.e.* from foetal relics). Normally young chorionic villi possess a double covering: the **superficial**, consisting of a multinucleated plasmodium which buds, and in which no cell outline can be distinguished, and a **deep layer** of distinct cells with definite outlines (layer of Langhans). The superficial represents fœtal epiblast; the deep is a specialised stratum of connective-tissue corpuscles of the stroma

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[1] Whitridge Williams, *Johns Hopkins Hospital Reports*, vol. iv. no. 9, 1895, p. 461.

[2] Mayer, *Virchow's Archiv*, vol. lxvii. See also Delbet, *Traité de Chirurgie*, 2nd ed. vol. viii. p. 186, 'Déciduomes'; Hartmann and Toupet, *Ann. de Gynéc. et d'Obstét.* 1895, t. i. p. 284.

[3] Neumann, 'Ueber Blasenmole und malignes Deciduom,' *Verhandl. der Deutschen Gesellsch. für Gynäk.* 1897, p. 304.

[4] Eden, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 153.

[5] Helme, *Trans. Royal Soc. Edin.* 1889.

(Eden). It is possible that the superficial plasmodial layer of the villus is the origin of such syncytial masses as described in deciduoma (*i.e.* fœtal epithelium); on the other hand, syncytium is maintained by some to be maternal (*i.e.* metamorphosed uterine epithelium).[1] The question arises, Is it necessary to draw on either theory for the occurrence of these plasmodial masses at all?

Kanthack and Eden, in examining sections of sarcoma of the testis, found plasmodial masses exactly like those said to be characteristic of deciduoma, formed by the active growth and fusion of sarcoma cells, accompanied with vacuolation and hæmorrhage.

The syncytial theory, therefore, of the plasmodial masses[2] seems unnecessary, and the great majority of cases of so-called deciduoma malignum ought really to be classed as rapidly growing sarcomata.[3] The connection of the growth with a preceding pregnancy is not proved in every case, nor its development from structures which can be definitely recognised as fœtal relics.

As regards pregnancy, such a sarcoma might be modified by its concurrent stimulus, but there is no proof that pregnancy, abortion, or the retention of decidua or chorionic villi, have caused the malignant growth (Kanthack). Bland-Sutton[4] holds that it is a sarcoma arising in decidual tissue. These probably represent the views of most English gynæcologists, but abroad, in France and Germany, the origin of such new growths from fœtal relics is held by many,[5] *i.e.* that they are derived from tissues, fœtal epithelium or epiblast, living in maternal tissues after the death of the ovum, a new pathological entity according to English observers; if so, then, such growths are not deciduomata (sarcomata) but chorionic carcinomata or epitheliomata.

Vesicular degeneration of the chorion is the most suggestive condition as mentioned before, and it is in connection with this that the required proof is most likely to be found.[1] In fact in Germany to such hydatid mole degeneration the name of 'Chorio-epithelioma benignum' is given, while if the remains undergo malignant degeneration it is called a 'Chorio-epithelioma malignum.'[2]

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[1] NOTE.—In either case the syncytium would be epithelial in origin, and the growth would therefore be a carcinoma, not a sarcoma.

[2] Plasmodial masses are characterised by
- (*a*) Absence of cell outline;
- (*b*) Deeply-staining nuclei;
- (*c*) Vacuolation.

[3] Eden, Kanthack, *Trans. Obstet. Soc. Lond.*, *loc. cit.*, discussion, pp. 162, 173.

[4] Bland-Sutton, *Trans. Obstet. Soc. Lond.*, *loc. cit.*, discussion, vol. xxxviii. p. 179.

[5] Delbet, *Traité de Chirurgie*, 12th ed. vol. viii. p. 186; Cullen, *Cancer of the Uterus*, p. 597.

Adenoma Malignum.[3] (Figs. 20, 21, 140.)—It cannot be said that this can be exactly considered as a distinct form of disease, yet the subject is of great interest, and from time to time reports of cases come before us tending to the conclusion that it may be a distinct form of cancer. The question is not settled. The condition generally occurs in old women past the climacteric, as nodules usually developing in the body of the uterus. Cases are very rare in the cervix. Sinclair[4] reports a case in a woman of 57, in which he says the characteristic glandular appearance of malignant adenoma could be very definitely made out. Williams,[5] quoting Matthews Duncan, describes such a case in a woman of 52, in which a tumour occurred in the fundus, composed of glands lined with a continuous stratum of epithelium, with much infiltration and hæmorrhage into the connective tissue. There was much uterine hæmorrhage, discharge, and passage of pieces of the growth. It recurred in five months, and although unlike a carcinoma in structure, resembled it clinically. The changes in the glands in adenoma are of two kinds: the cells may retain pretty much the character of epithelium of the glands in health, or they may be somewhat smaller and regularly formed, or they may be larger[6] (fig. 140). Glandular growths limited to superficial tissues lead us naturally to glandular growths which are not thus limited, and which involve deep structures, affect the whole thickness of the cervix, and invade

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[1] Fothergill, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 177, 'Discussion on Deciduoma.' Also Champneys, 'Some unusual Cases of Hydatid Mole,' *Practitioner*, January 1896, p. 15, Case 3, p. 20; Apfelstedt and Aschoff, *Arch. für Gynäk.* vol. i. p. 96.

[2] L. Fränkel, *Trans. Obstet. Soc. Lond.*, vol. xli. 1899, p. 336.

[3] Ruge and Veit, *Zeit. für Geb. und Gynäk.* 1882, vol. vii. p. 170. See Delbet, *Traité de Chirurgie*, 2nd ed. vol. viii. p. 198, 'On Benign and Malignant Adenoma;' Cullen, *Cancer of the Uterus*, 1900, p. 304; Gebhard, *Zeit. für Geb. und Gynäk.* 1895, vol. xxxiii. p. 443; Routh, 'Malignant Papilloma of the Uterus,' *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 5; Eden, 'Specimen of Adenoma of the Uterus,' *ibid.* vol. xlii. 1900, p. 2 (see figs. 20, 21, in this book); Roberts, *ibid.* vol. xli. p. 267, and *Brit. Med. Journ.* vol. ii. 1900, p. 1109.

[4] Allbutt and Playfair, *op. cit.* p. 730.

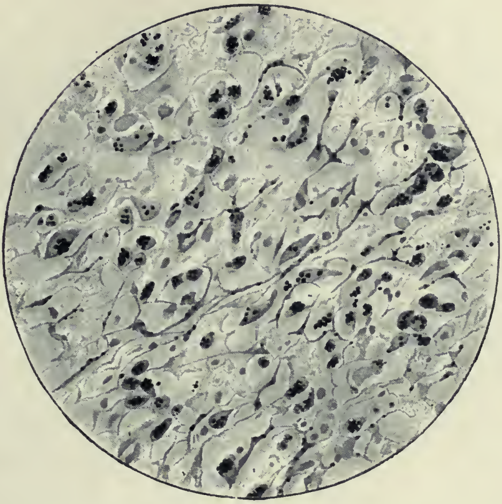
[5] Sir J. Williams, *Cancer of the Uterus*, p. 100.

[6] *Id. op. cit.* p. 104.

Fig. 139.

DECIDUAL CELLS

From a Five Weeks' Ovum, removed in a Pregnancy which followed Puerperal Eclampsia. (*Ad naturam.*)

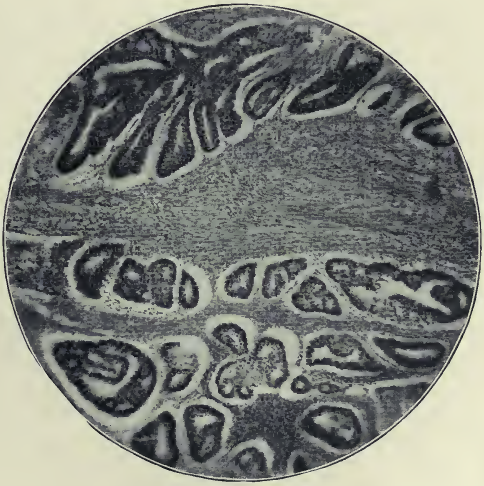


Decidua vera, showing deposit of fat in the nuclei and to a slight extent in the reticulum. Note the wide empty spaces.

(Eden and Griffith, 'Trans. Obstet. Soc. Lond.' vol. xli. plate ii.)

Fig. 140.

ADENOMA MALIGNUM OF THE BODY OF THE UTERUS.



Section of a portion of the muscular wall of a uterus, showing extensive infiltration by a growth of a malignant character.

The type of the growth imitates more or less the glands of the body of the uterus; the cells are arranged in acini, but the irregular and extensive infiltration of the deeper layers of the uterus by the growth, and the atypical proliferation of the cells lining the acini, make the growth pathologically a malignant one.

(Section by Corrie Keep. Microphotograph by C. H. Cosens.)

the connective tissue beyond; these adenomata possess malignant properties.[1]

Sinclair reports a case quoted by Länderer which he says was clearly a case of adenoma malignum[2] in a woman of 48: the uterus was removed after five years of hæmorrhage, and one of treatment, for a recurring tumour of the fundus. There were lying throughout the whole muscular layer of the uterus islands of a tissue exactly resembling the mucous lining in structure, consisting of gland-like coils of tubes, with long, narrow, closely-planted cylindrical epithelium. Recovery took place. Still, all activity of the endometrium in post-climacteric women is very suspicious of malignancy, and Sinclair[3] says all post-climacteric polypus or fungus of the endometrium of the body of the uterus is to be regarded as adenoma malignum.

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[1] Sir J. Williams, *op. cit.* p. 42.

[2] Allbutt and Playfair, *op. cit.* p. 732; Länderer, 'Adeno-carcinom des Corpus Uteri,' *Zeit. für Geb. und Gynäk.* 1892, vol. xxv. See also Ruge and Veit, *ibid.* 1882, vol. vii. p. 170. See also Cullen, *op. cit.* p. 304, who quotes Gebhard's cases.

[3] *Op. cit.* p. 734.

CHAPTER XVI

DISEASES OF THE BLADDER.

Normally[1] the empty bladder lies below the pelvic brim; when relaxed, its upper surface is concave, and on section its walls are thick and its mucosa wrinkled, the cavity being a mere curved slit with its concavity forwards. When moderately full, the bladder becomes rounded, and its upper surface lies above the brim; if more distended it becomes egg-shaped, being larger below; in severe distension the fundus may reach the navel.

The superior surface is covered with peritoneum and is in relation with the uterus. The reflections of the peritoneum from the bladder form the false ligaments, two behind (the utero-vesical), two laterally, and one in front (the superior). The summit is connected to the abdominal wall by a mesial cord, the urachus, the stalk of the allantois.[2]

Laterally,[3] are the obliterated hypogastric arteries, connected with the superior vesical arteries, which run forwards and upwards to the navel. Below these vessels, the bladder is in relation with the para-vesical connective tissue. Anteriorly, the bladder is in relation to the back of the symphysis, and the loose areolar tissue in that region. Posteriorly, the bladder is in relation with the cervix and upper part of the vagina, separated by cellular tissue. Generally, in the erect position, the urethra is the lowest part of the bladder. A bladder sound on an average passes 4½ inches when the viscus is empty. It is doubtful whether the capacity of the female bladder is greater than that of the male.

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[1] Clarence Webster, *Diseases of Women*, p. 58.

[2] Doran, 'A Case of Cyst of the Urachus, with Notes on Urachal and so-called llantoic Cysts,' with literature, *Trans. Med. Chir. Soc. Lond.* vol. lxxxi. p. 301.

[3] Clarence Webster, *op. cit.* p. 59.

Hunner and Lyon,[1] in carrying out some experiments on the capacity of the female bladder in the living woman placed in the knee-breast position, found that the average atmospheric distension capacity of the healthy bladder was 303 cc. (10½ oz.), and that the average fluid capacity was 429·7 cc. (15 oz.), or ⅓ greater, probably owing to the greater power of fluid pressure. Anything above this gave rise to pain. On anæsthesia being employed to eliminate pain or spasm, the results were :—

	Atmospheric capacity	Fluid capacity
With anæsthesia	306·7 cc.	449·6 cc.
Without „	295·0 cc.	387·1 cc.
	303·0 cc.	429·7 cc.
	Average 25 cases.	Average 22 cases.

Comparing nulliparous women with parous, it was found that the bladder capacity was slightly greater in the former, as probably childbirth and scleroses were causes of diminution of bulk.[2]

Measurements of the internal dimensions of the normal bladder were also taken from the internal orifice of the urethra. They were as follows :—

a. To the vertex of the bladder = 7·14 cm. (3 in.).
b. To the posterior wall = 5·77 cm. (2·3 in.).
c. To the left lateral wall = 6·70 cm. (2·7 in.).
d. To the right lateral wall = 5·92 cm. (2·36 in.).

The measurements were slightly greater in nulliparous women. The same observers found that the bladder tends to be displaced rather to the left than to the right side of the pelvis, as the rectum is oftener on the right side of the pelvis than the left in women, and the uterus encroaches rather on the left upper quadrant of the bladder.

Distension of the bladder caused it to assume an ellipsoidal form, flattened somewhat in the antero-posterior diameter, while the transverse, which is always the greatest diameter, is further increased.

Measurements of the healthy urethra in the female in seventeen cases = 3·3 cm. (1·3 in.) in average. The shortest measurement was 2·7 cm. (1·08 in.), and the longest 4·2 cm. (1·7 in.). Any distension of an inflamed bladder gives rise to great pain. Generally four ounces in such cases is the amount that can be borne; more than this causes distress.

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[1] 'Mensuration and Capacity of the Female Bladder,' Hunner and Lyon, *Johns Hopkins Hospital Bulletin*, vol. x. No. 105, p. 227.

[2] Hunner and Lyon, *op. cit.* p. 227.

The **Muscular Wall** consists more or less of three layers: an outer longitudinal, a middle thin reticulated layer, and an inner longitudinal.[1]

The Mucous Membrane is pink in colour, soft and rugose, except when the bladder is distended, and is loosely attached to the muscular wall except at the trigone of the bladder. It consists of stratified transitional epithelium; the rugose condition seen in the empty bladder is due to the wrinkling of the mucosa and also to the ridges formed by the underlying muscular fasciculi.

The **Trigone** is the triangular area, bounded by lines each about 1$\frac{3}{8}$ inch long, joining the urethral and ureteral orifices; here the mucosa is smooth, and is firmly attached to the muscular coat. The ureteral orifices form oval slits looking forwards and inwards. Joining the ureteral orifices is a sort of curved ridge or prominence due to a submucous muscular band, in the middle of which is a small eminence sometimes called the *uvula vesicæ*.

In **Infancy** it is well to remember that the bladder is almost wholly an abdominal organ, and above the symphysis. It gradually sinks as the pelvis develops, but is partly abdominal even up to the sixth year.[2]

Absence of the Bladder.[3]—Only a few instances of this are on record. When it occurs, the ureters may open into the urethra, rectum, or vagina, or on the surface of the abdomen, generally in the median line.

Ectopia Vesicæ.[4]—This is caused by a failure in development of the anterior wall of the bladder and of the abdominal wall in front of the bladder; the posterior wall of the bladder projects at the hypogastrium, where it is continuous with the anterior abdominal parietes. The condition is much more common in the male than the female. It is due to a failure of the abdominal laminæ to unite in early fœtal life; the existence of epispadias, the absence of union of the symphysis, and other malformations of the genital organs, are other points in favour of this view.

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[1] C. Webster, *op. cit.* p. 59.

[2] C. Webster, *op. cit.* p. 60.

[3] Henry Morris, article, 'Diseases of the Female Bladder and Urethra,' Allbutt and Playfair, p. 930.

Champneys, *St. Bartholomew's Hospital Reports*, vol. xiii.

Less degrees of the defect are sometimes seen as a furrow in the anterior abdominal wall, dividing the clitoris into right and left halves and separating the labia; sometimes an indication of this deformity exists as a narrow furrow over the symphysis reaching up to the navel.

Failure of the urachus to close high up leaves a vesico-umbilical fistula[1] through which urine escapes; or, instead of merely a fistula, a fissure may extend into the upper part of the bladder. In advanced cases more and more of the mucous membrane is exposed till the whole of the anterior wall of the bladder is wanting as above described. When the pelvis is widely 'split' in front, a fibrous band sometimes unites the pubic rami, which may be one and a half to six inches apart; the recti are separated, and the navel may be absent.[2]

On inspection, ectopia vesicæ appears as a rounded mass just above the position of the vaginal orifice, low down in the pubic region; its size varies, and it is generally widest in its transverse diameter; the surface is irregular, wrinkled, and looks like a 'spongy mass of excoriated flesh.'[3] It is always moist with urine and a slimy discharge; frequently the odour from such patients is horrible. The surface is very sensitive and bleeds readily. The lower part is always moister and more vascular than the upper. Here two small rounded projections exist, representing the orifices of the ureters. Urine can be seen escaping from them, not drop by drop, but by a sort of feeble irregular ejaculation.[4]

Around the edges of the surface the skin blends insensibly into the mucous membrane. Sometimes little patches of epidermis may be situated on it.

The mucous surface may extend upwards to the umbilicus, or the umbilicus may be absent.

The separation of the labia majora and minora, and the two sides of the clitoris, has already been pointed out. The vagina may be a mere slit antero-posteriorly, and in some cases the sex of the infant is doubtful.[5] The anus is often placed farther forward than normal; the sacrum is also far forward, and the antero-posterior diameter of the pelvis diminished, while the lateral diameters are wide. The perineal muscles are ill developed and there is no sphincter to the bladder. The ureters, according to Morris,[1] dip down into the pelvis before turning up towards the kidneys, and are frequently elongated and dilated.

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[1] Howard Kelly, *Operative Gynæcology*, vol. i. p. 319; see also Doran, *Trans. Med. Chir. Soc. Lond.* vol. lxxxi. p. 301.

[2] Henry Morris, *op. cit.* p. 930.

[3] H. Kelly, *op. cit.* vol. i. p. 319.

[4] Henry Morris, *op. cit.* p. 930.

[5] *Ibid.*

Patients with ectopia vesicæ are rarely healthy; their sufferings and discomforts are constant. The bladder is easily irritated and excoriated, and the surrounding skin areas inflamed by the decomposing urine; the inflammation may extend up the ureters and end in suppurative pyo-nephrosis and death.

As a rule, sexual appetite is absent and conception rare, but it has occurred; in some cases delivery has been [2] difficult, and prolapse of the uterus has followed it.

Malformations of the uterus and vagina occur, and hernia is common. Morris [3] says that the ileum may end in the bladder. Other deformities, such as club foot, anencephalus, and spina bifida, may exist.

Ectopia vesicæ is rare. According to Neudorfer, quoted by Morris, it occurs twice in 100,000 infants; nine-tenths of the cases die within a few days of birth, but some cases have lived to the ages of forty, fifty, and even seventy years.[4]

An extroverted bladder may become carcinomatous.[5]

Displacements of the Bladder.—As previously pointed out, in marked distension the female bladder rises into the abdomen, with its greatest diameter vertically, and resembles a cyst, and serious errors may be made in diagnosis if the passage of a catheter be neglected. Sometimes the bladder, when distended, may not be in the middle line, but inclined to one side or the other. Upward displacement apart from distension is seen with pregnancy and fibroid tumours; in the latter, the displacement and distortion of the bladder are most remarkable, and at operations every care should be taken beforehand to discover its limits with a bladder sound. Smaller tumours jammed in the pelvis easily lead to distension of the bladder upwards, and with any abdominal tumour fulness of the bladder should always be excluded.

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[1] Henry Morris, *op. cit.* p. 931.

[2] Litzmann, *Arch. f. Gyn.* vol. v., and Gusserow, *Berlin. klin. Wochenschr.* 1879, No. 2; H. Kelly, *op. cit.* p. 319.

[3] Henry Morris, *op. cit.* p. 931.

[4] The author has recently had under his care at the Samaritan Free Hospital a well-marked case of ectopia vesicæ in a woman of 36. She also had prolapse of the uterus and rectum. There was apparently only one kidney, which was in a condition of hydronephrosis. Only one ureteral opening could be discovered on the exposed bladder surface. She was in fair health.

[5] See H. Kelly, *op. cit.* vol. i. p. 320, fig. 204.

Downward displacements of the bladder are seen in descent of the pelvic floor associated with prolapse of the womb; the condition (cystocele) has already been described. Owing to the firm attachment of the vaginal wall to the bladder, it is very rare for the vagina to descend without the bladder, though Kelly[1] reports a case where the bladder was detached from the vaginal wall and uterus in a prolapse, and remained entirely inside the pelvis.

Eversion of the Bladder through a dilated urethra is very rare (see Diseases of the Urethra). It occurs generally in children. The bladder has rarely been seen in the sacs of inguinal, femoral, and obturator herniæ.

Foreign Bodies.—These may occur in the bladder:

(*a*) Introduced through the urethra.

(*b*) They may form in the bladder itself as calculi, or descend from the kidneys.

(*c*) They may reach the bladder from the exterior by perforation of the wall, either by injury or ulceration, with the formation of a fistula.

Stone in the bladder will be considered separately later.

By the urethra a very large variety of foreign bodies have been introduced. If of some size, they occupy a transverse position between the summit and the neck of the bladder rather than the neck itself.[2] Very long foreign bodies may take up almost a vertical position.

Such foreign bodies may remain entirely quiescent, or they may give rise to cystitis, and after a time ulceration and perforation occur, or a peri-vesical abscess may form, and the body be discharged with the pus; or the foreign body, having perforated the bladder wall may remain partly in the bladder and partly in the peritoneal cavity.

Foreign bodies become encrusted with phosphates, and are then often the nucleus of a stone. The deposit generally takes place upon the largest part of the foreign body near its middle, and proceeds towards the extremities, but the ends are never encrusted.[3]

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[1] H. Kelly, *Johns Hopkins Hospital Reports on Gynæcology*, vol. ii. 1892, p. 311.

[2] Henry Morris, *op. cit.* p. 949.

[3] Henry Morris, p. 949; see also H. Kelly, *op. cit.* vol. i. p. 328, fig. 205.

In cases of injury, violence, or gunshot wounds, bullets, pieces of bone or clothing, may be found in the bladder.

By ulceration dermoid cysts have opened into the bladder, discharging hair, teeth, fat, and other highly irritating structures (see 'Ovarian Dermoids,' p. 211).

Bones from an extra-uterine gestation sac have got into the bladder in this way, pelvic abscesses around a foreign body, and hydatid cysts have burst into the bladder: vaginal pessaries have ulcerated through the vesico-vaginal septum; fæcal matter and intestinal worms have entered from the bowel.

Stone in the Female Bladder.—This is much rarer than in the male, owing to the shortness and dilatability of the female urethra, for small calculi are easily passed; besides this, Morris says that gravel and gout are less frequent in women than in men.[1]

Beyond the foreign bodies mentioned above as possibly giving rise to the nucleus of a calculus, decomposition of the urine and cystitis are most likely to lead to the precipitation of the ammonio-magnesium phosphates. This may take place spontaneously; much oftener it occurs on some small concretion which has descended from the kidney, in which case the nucleus is generally uric acid. Foreign bodies or the surface of vesical neoplasms are encrusted in a similar way.

Sometimes even fairly large stones may be passed by the urethra; larger ones generally make their way into the vagina by ulceration of the vesico-vaginal septum. The most serious results from stone are cystitis and suppurative pyelo-nephritis, leading to a fatal event.

Cystitis in the female is perhaps less common than in the male, due probably to the length and retaining influence of the male urethra, and to the frequent troubles that arise in men with diseases of the prostate and stricture.[2]

Cystitis may be acute or chronic.

Acute Cystitis.—Every case is probably due to septic infection,[3] and the continuance of the disease depends upon the continued action of one or other of the various pathogenic micro-organisms. The commonest form of infection occurs through the urethra in the passage of an unclean catheter: gonorrhœa, tuberculosis, calculus, and vesical new growths are other less common causes.

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[1] Henry Morris, *op. cit.* p. 956.

[2] H. Kelly, *op. cit.* p. 360.

[3] Henry Morris, *op. cit.* p. 936.

Morris[1] points out the intimate relationship between the uterus and bladder in the female, and notes the remarkably free vascular communications which exist between them, especially between the vesical and anterior uterine veins, and shows that the bladder shares in the peculiar vascular changes connected with menstruation.

These changes are perhaps best seen in connection with pregnancy, in the irritability of the bladder before the uterus has reached any appreciable magnitude. Further, the displacement of the bladder by the retroverted gravid uterus and by the full-term pregnant uterus, and the risks of bruising in difficult labour, all tend to render the relations between uterus and bladder one of interest in connection with cystitis. Lastly, in the puerperal period, too great care cannot be taken to avoid cystitis by stringent asepsis.

The bladder is said to be infected from the kidney in some cases as in pyelitis and pyelonephrosis, especially of a tubercular nature, but the subject is a little difficult of proof as they may all be part of one primary infection.

It is possible that bacteria may also enter the bladder directly[2] from inflammatory areas in the neighbouring organs, such as diseased Fallopian tubes, the blood itself remaining sterile, and Kelly[3] brings forward some evidence that in a few cases direct infection of the bladder may take place from the intestines, but even then it is difficult to eliminate blood infection, or infection from the kidneys.

Much of such evidence is based on experimental closure of the urethra and rectum in animals[4] which occasions a true cystitis. The infection may of course come from perforation or rupture of the bladder, or from rupture of purulent collections from other viscera, such as a tubal, ovarian, or perityphlitic abscess, from inflamed ovarian cysts, and also from certain new growths of the bowel involving the bladder secondarily. Air and fæces may escape into the bladder,[5] though this is not necessarily attended with cystitis: again, new growths in the bladder, cancer, tubercle, polypi, and villous tumours are liable to set up cystitis.

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[1] Henry Morris, *op. cit.* p. 936.

[2] H. Kelly, *op. cit.* p. 361.

[3] Reymond, 'Annales des Maladies des Organes gén.-urin.,' Ap. 1893, quoted by Kelly, *Operative Gynæcology*, vol. i. p. 361.

[4] Wreden, *Arch. de Sci. Biologiques, St. Pétersbourg*, vol. ii. p. 5, 1894, quoted by Kelly, vol. i. p. 360.

[5] Harrison Cripps, 'On the Passage of Air and Fæces from the Urethra,' 1888, p. 5. Out of sixty-three cases of entero-vesical fistula collected, two were traumatic, nine cancerous, forty-five inflammatory; unascertained, seven.

Retention of urine in any case is a serious predisposing factor.

Micro-organisms in Cystitis.—Cystitis is due to the decomposing and infective power of various bacteria, of which the alkaline and ammoniacal urine is a secondary result.

Many different organisms have been isolated. Howard Kelly[1] gives an excellent summary of the most recent papers on this subject by Doyen, Rorsing, Krogius, Albarran and Halle, Melchior, and others. Among the organisms most commonly isolated are the bacillus coli, the gonococcus, the streptococcus pyogenes, the staphylococcus pyogenes albus, citreus, and aureus. The bacillus of typhoid and tubercle, the urobacillus liquefaciens, and several varieties of proteus have also been found.

Kelly[2] summarises the matter thus:—

1. That cystitis is always due to the presence of bacteria.
2. That the mere presence of bacteria is insufficient to cause a cystitis, a further predisposing cause being necessary.
3. That there are various modes of entry for bacteria: through the urethra, through the ureter from the kidney directly, from inflammatory areas in the uterus or Fallopian tubes, and probably from the rectum under like conditions; still another probable avenue of entrance is through the blood.
4. That under favourable conditions any pathogenic organism may give rise to cystitis.

Pathological Results of Cystitis.—The initial change in cystitis is infection of the blood-vessels of the mucosa near the neck of the bladder; this extends, and the whole mucous membrane swells and becomes a bright red colour. The epithelium at first swells, and the changes are accompanied by catarrh, and admixture of the urine with serum, mucus, and epithelial *débris*. Later, deeper layers of the bladder-wall are affected; the rete mucosum, and sometimes the muscular coat, being infiltrated with small inflammatory cells. In worse cases ulceration may ensue, and a purulent catarrh is established; occasionally portions of the mucosa slough, and large pieces may be passed. False membranes sometimes form in the same way, or abscess and gangrene may result. Acute cystitis in the puerperal period is sometimes severe and prolonged, and is a very dangerous condition.

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[1] H. Kelly, *op. cit.* vol. i. pp. 362, 363.

[2] *Op. cit.* p. 364.

Chronic Cystitis is characterised by the formation of much stringy viscid mucus, and by alkalinity of the urine. The mucous membrane of the bladder is swollen and thickened, with the prominent veins and areas of congestion and submucous extravasation;[1] its colour is dark grey, with patches of purple or dusky red. Phosphatic deposits often occur, and occasionally there are ulcers on the trigone. The urine in such a case is turbid and alkaline, with a foul ammoniacal smell; it is mixed with mucus, pus, and blood, and, on standing, deposits crystals or triple phosphates. The bladder itself is often contracted and thickened.

The false membranes in cystitis[2] are generally composed of fibrinous material, containing in their substance leucocytes and epithelial cells, and are sometimes encrusted with phosphates. Membranes formed in very acute cystitis, and in cystitis of the puerperal period, may even invade the ureters and renal pelves; in other cases the false membrane or cast may be made up entirely of many layers of epithelium, and in the gangrenous cases may be mixed with some of the constituent parts of the mucosa, which has been more or less destroyed.

The constitutional effects of chronic cystitis may not be marked for some time, but at length patients become feeble and sallow, and lose flesh. It is accompanied by a dry skin, furred tongue, poor digestion, and a hectic temperature; at length chronic pyelo-nephritis ensues, and this may be fatal. In some cases an acute attack of this nature may kill the patient.

Tubercular Disease of the Bladder.—Tubercle affects the bladder generally, along with other parts of the genito-urinary tract. It may commence in the bladder or the kidney. It may be met with in children and young adults. Morris[3] says it is most prone to occur during the active period of the sexual organs, and that gonorrhœal discharges and infective cystitis in persons with this proclivity are apt to pass into tuberculous disease, and that it is very rare for the bladder to be the only part of the genito-urinary apparatus affected at the time of death.

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[1] Bowlby, *Surgical Pathology*, 2nd ed. p. 400.

[2] Henry Morris, *op. cit.* p. 940.

[3] Henry Morris, *op. cit.* p. 943.

The morbid changes in the bladder are at first the growth of small grey tubercles in the mucosa, most numerous about the trigone; these after a time break down, and form circular superficial ulcers without any induration. Gradually, by an extension of this process, the mucous membrane is destroyed, and general cystitis is set up. Large cheesy masses like those met with in the kidneys and tubes do not occur. The ulceration in the later stages may be very extensive and deep, leading to perforation into such cavities as the rectum or vagina, or, externally, to fistulous openings in the perineum, or above the symphysis.[1] The urethra may even be involved.

The ureters show similar changes, and can sometimes be felt through the vagina as thick tubes, the thickening and ulceration sometimes obstructing the passage of urine. For the changes in the kidney, works on Surgical Pathology must be consulted. Slight hæmaturia may occur in tubercular cystitis either in the early or late stages. Sometimes the pain in this disease is agonising, preceding, accompanying, and following micturition, and nothing gives relief till perhaps ulceration (leading to perforation) occurs, and the bladder is thus in a way drained. The urine may be fairly clear in early stages; later it is like that of chronic cystitis. Tubercle bacilli may be found in the early stage, but not when there is much pus.[2] The uterus and tubes may show evidence of tubercular disease as well.

Injury to the Bladder. Urinary Fistula.—Tears of the bladder may be caused by direct violence from without, possibly in some cases from violent muscular efforts. Sloughing may occur as the result of a difficult labour.

The pressure of a retroverted gravid uterus has caused gangrene of the walls of the bladder in several instances.[3]

In connection with diseases of women, rupture itself is a rare event, but the occurrence of urinary fistulæ is not very uncommon.

A common cause of communication between the bladder and vagina is cancer of the cervix,[4] destroying the vesico-vaginal septum, and in advanced cases the vagina may be converted into a mere cloaca.

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[1] Henry Morris, *op. cit.* p. 943.

[2] Henry Morris, *op. cit.* p. 944.

[3] Henry Morris, Allbutt and Playfair, *op. cit.* p. 947.

[4] Herman says it occurs in one-sixth of all cases of cancer: *Diseases of Women*, p. 363.

Mechanical injuries during parturition are the next important set of causes. There may be direct tears of the bladder by instruments, but this is rare, and in by far the greater number of cases the fistula results from sloughing. Nineteen out of twenty fistulæ are caused in this way.[1] The symptoms do not come on at once as with direct tears, but are postponed till the slough separates. The sloughing is caused by the continuous compression of the soft tissues between the child's head and the pelvic bones, and in contracted pelvis it is most likely to occur either at the promontory of the sacrum or the symphysis. The exact situation of the sloughing depends often upon the extent of the dilatation of the cervix, and large portions of the cervix may slough as well as the bladder,[2] and even the ureters may be involved. Fistulæ involving the cervix are rarer,[3] and no uretero-cervical fistula has ever been dissected after death.[4] Nearly all fistulæ of this nature are caused, not by the use of forceps in difficult labour, but because they are not used soon enough.

The result of a vesical fistula is incontinence of urine, which is more or less complete, though sometimes a little urine can be retained. There is usually cystitis as well, the urine is ammoniacal, and the odour of decomposing urine is very apparent in these patients; they are always wet, and great irritation and distress results from inflammation of the labia and skin.

Openings into the bladder from congenital deformities such as hypospadias and extroversion have already been considered.

Very much less frequent causes of urinary fistulæ are necrosis of the bladder itself in severe inflammation, or ulceration from long-continued pressure of a pessary.

Occasionally fistulæ, either of the bladder or ureter, occur after operations for removal of the uterus by the vagina.

The chief **varieties of urinary fistulæ** are:

1. Vesico-vaginal.

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[1] Herman, Allbutt and Playfair, 'Injuries in Parturition,' *op. cit.* p. 436.

[2] Herman, *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 244.

[3] Cervical fistulæ form in 8 per cent. of vesical fistulæ after delivery. Neugebauer, *Arch. f Gyn.* Bd. xxxiv., quoted by Herman, *Diseases of Women*, p. 688.

[4] Herman, *op. cit.* p. 437.

2. Cervico-vesical (vesico-uterine).[1]
3. Vesico-cervico-vaginal.
4. Urethro-vaginal.
5. Uretero-vaginal.

Nocturnal Incontinence.—This is a most troublesome affection, generally seen in children, but in most cases it disappears at puberty.

Some children have incontinence at night whose urinary functions during the day are quite normal. This, according to Morris,[2] constitutes a large class of such cases, and the incontinence is of a 'psychopathic' (mental) origin, in fact a neurosis. In another class of cases there is also diurnal incontinence, and if there is not immediate relief the child wets its clothes. This is due to either irritation of the spinal cord, intestines, or genito-urinary apparatus; contracted meatus, oxaluria, lithæmia, and intestinal worms play an important part in it. In another class, there is incontinence night and day,[3] and the children make no effort to retain their urine at all. This form, according to Morris, is due to defective contractile power in the urethral sphincter, or urethral insensibility.

Incontinence during epileptic seizures must not be forgotten.

Incontinence of Urine.—In this condition the patient cannot retain any urine, and as it flows into the bladder from the ureters it escapes externally through the opening in the bladder wall ('true incontinence'). This must be carefully distinguished from the overflow from a full bladder, sometimes called 'false incontinence.' Urinary fistulæ have already been described.

The chief **causes** of incontinence are:[4]

a. Damage to the urethra or bladder during labour or operations, or by some accident.
b. Sloughing from inflammation, labour being the most common cause.
c. Breaking down of malignant disease.
d. Congenital malformation.

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[1] Champneys, 'A new Operation for Vesico-uterine Fistula,' *Trans. Obstet. Soc. Lond.* vol. xxx. 1888, p. 348.

[2] Henry Morris, Allbutt and Playfair, *op. cit.* p. 934.

[3] Henry Morris, *op. cit.* p. 935; see also *Id.* 'Functional Disturbances of the Bladder,' *op. cit.* p. 932.

[4] Herman, *Diseases of Women*, p. 686.

Retention of Urine.[1]—The chief causes need only be mentioned here, as many of the conditions themselves have been described elsewhere.

- *a.* Hysteria.
- *b.* Retroversion of the gravid uterus.
- *c.* Fibroids or retro-uterine swellings, such as an ovarian tumour, serous perimetritis, retro-uterine hæmatocele, a malignant growth, extra-uterine gestation, or hydatid cyst.
- *d.* The second stage of labour.
- *e.* The puerperal state, from lack of expelling power, neurosis, or injury to the urethra.
- *f.* Nervous diseases, such as tabes dorsalis, disseminated sclerosis, and paraplegia.

Painful Micturition.—The causes of this may be slight or severe; most of them have already been described. They are (Herman) :—

- *a.* Slight.
  - 1. Concentrated urine.
  - 2. Pelvic inflammation.
  - 3. Gonorrhœa.
- *b.* Severe.
  - 1. Caruncle.
  - 2. Diseases of the meatus or urethra itself.
    - *a.* Chronic congestion.
    - *b.* Abscess or suppurating cysts.
    - *c.* Tender red spots in the urethra.
  - 3. Diseases of the bladder.
    - *a.* Fissure of the neck of the bladder.
    - *b.* Cystitis, from whatever cause.

Too frequent Micturition (irritable bladder).—The causes, according to Herman,[2] may be :

- *a.* Those in which there is no local disease appreciable by the ordinary methods of examination.
  - 1. Pregnancy.
  - 2. Nervous disease.
  - 3. Smallness of the bladder.
  - 4. Over-distension of the bladder.

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[1] Matthews Duncan, *Clin. Lect.* 4th ed. p. 83; Herman, *Diseases of Women*, p. 679.

[2] Herman, *Diseases of Women*, p. 643.

        5. Excessive quantity of urine.
        6. Nocturnal enuresis.
    b. Reflex irritation of the bladder.
        1. Due to some local cause, such as tampons in the vagina, pessaries, etc.
        2. Due to descent.
        3. Dislocation of the urethra by tumours, pregnancy, or prolapse.
    c. Local disease.
        1. Vulvitis.
        2. Vaginitis.
        3. Stricture of the urethra.
    d. Bladder or renal disease.
        1. Cystitis.
        2. Bright's disease.
        3. Pyelitis or pyelo-nephrosis, from whatever cause.

Tumours of the Bladder.—Tumours of the bladder comprise both innocent and malignant growths. The former include papilloma, myxomatous polypi, fibrous polypi, and myomata, while the latter include carcinoma and sarcoma. Tumours of the bladder occur oftener in males than females, and in either sex malignant tumours preponderate over innocent.

The following table is given by Morris,[1] showing their relative frequency:

—	Males	Females	Total
Cancer	43	16	59
Sarcoma	5	1	6
Fibroma	1	1	2
Papilloma (villous)	21	2	23
	70	20	90

Nearly all new growths of the bladder occur about the trigone and near the orifice of the ureters. Benign tumours are often polypoid and single, while the malignant ones have a large base with much infiltration, and may be multiple.

The papillomata are the most common of the innocent tumours, and are generally of the 'villous' type, being made up of delicate branched papillæ or processes,[2] each of which is found to consist of a central stalk of delicate connective tissue in which are embedded many round and oval cells. The processes are very vascular, containing a central vessel, the surface being covered with oval or fusiform epithelial cells. The whole growth may be either sessile or pedunculated, or occur in tufts. They may give rise to alarming hæmorrhage, and if the long villous processes which float in the urine get carried into the urethra, they cause great pain.

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[1] Henry Morris, *op. cit.* p. 951. [2] Bowlby, *Surgical Pathology*, 2nd ed. p. 402.

Another variety of papilloma[1] is firmer, though it has a villous type. It may be single or multiple. It is generally rounded in shape, not larger than a walnut, and is often sessile.

Myxoma and **Mucous Polypi** are rare, and are met with only in children. They are composed of myxomatous tissue, are soft in texture, and grow very rapidly. Morris thinks they may be be fibro-papillomatous or fibromatous, the cell portions of which have undergone mucoid degeneration. Bowlby compares them to the mucous polypi found in the nose.[2] They are often very numerous, covering the greater part of the mucous membrane, and they may extend into the urethra.

Fibrous polypi are described by Bowlby as being rare. They occur in children, and are liable to prove fatal by interfering with the passage of urine, and thus inducing kidney disease.

Fibromata[3] originate in the deeper portions of the mucosa or in the muscular layer, and are covered with the normal epithelium of the bladder. They may, like myxomata, be pedunculated; they only occur in adults. Myomata are very rare.

Malignant Tumours.—Epithelioma is often met with, and may start on any part of the bladder or in several places. In men, it is a disease of middle life, and possibly the same applies to women. The surface of the growth generally ulcerates before it attains any considerable size, and the whole tumour gets softened and macerated by the urine, the shreddy portions partaking of a villous type, but the tumour ultimately infiltrates the surrounding tissues, though its growth is generally slow.

Secondary malignant disease of the bladder may occur with cancer of the rectum, vagina, or uterus, infiltrating the coats of the bladder very deeply, forming large ulcerated surfaces. If the growth break down, fistula results.

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[1] Henry Morris, *op. cit. p.* 952.

[2] Bowlby, *op. cit.* p. 403.

[3] Henry Morris, *op. cit.* p. 952.

The involvement of the bladder and anterior vaginal wall in cancer of the uterus has already been pointed out (see also fig. 125).

Sarcoma is less common than carcinoma in the bladder. Bowlby[1] says they appear to behave as do sarcomata elsewhere, infiltrating, breaking down, and disseminating. They differ in appearance from the carcinomata in their greater bulk, they are fleshy on section, and their surface is not as ragged as in epithelioma.

Bilharzia hæmatobia, a parasite generally found in Egypt and South Africa, may attack the vesical veins,[2] and the ova penetrating sub-epithelial tissues of the bladder set up inflammation, ulceration and hæmorrhage (endemic hæmaturia). The heaped-up inflamed ulcerated mucosa of the bladder may simulate a tumour, and the whole interior of the bladder may be rough and villous.

The sexes of Bilharzia (a fluke) are distinct; the female worms are common, but the male rare; the ova are oat-shaped, with a terminal or lateral spine.

The pathological results of tumours of the bladder are important. Innocent tumours may exist for years and give rise to little trouble, and cystitis may be absent; others give rise to severe hæmorrhage, sometimes they obstruct the ureters or urethra, and may eventually set up secondary kidney disease.

Malignant tumours cause great irritation and severe cystitis, and calculous concretions form on the surface of the growth; they infiltrate and destroy the bladder-wall, or suppurative pyelonephritis brings about a fatal termination.

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[1] Bowlby, *op. cit.* p. 405.

[2] *Id. ibid.* p. 400.

CHAPTER XVII

ON DISEASES OF THE URETHRA

THE female urethra forms a slit in the pelvic floor parallel to the vagina, and is generally about one and three-eighths inches long. It is directed from the bladder downwards and forwards, sometimes with a slight sigmoid curve. Its anterior and posterior walls are in apposition except during micturition.[1] The external orifice is a vertical slit, with slightly projecting ridges, situated in the middle line at the base of the vestibule. Around the urethra the unstriated muscle is arranged in an outer circular and an inner longitudinal layer; among these is a considerable amount of connective and elastic tissue. There is also a rich vascular plexus in these layers, sometimes called the corpus spongiosum urethræ. Striped muscle exists outside it, mainly in a circular bundle around the upper end of the urethra and the internal sphincter. Another set of circular striped fibres exists at the lower end, blending with the anterior fibres of the sphincter vaginæ, from which it may be derived.[2]

The **Mucous Membrane.**—This is raised in longitudinal folds. One, especially marked along the posterior wall, is called the crista urethralis. The epithelium is transitional above, like the bladder; below, near the orifice, the cells are more flattened.

The mucous membrane is very rich in numerous glands and lacunæ, most marked in the upper part of the urethra. Clarence Webster says that in old women these may contain small brownish masses, like prostatic calculi in men.

Two small blind glandular ducts are sometimes found in the posterior wall, opening at the external orifice, called the ducts of Skene. They may be mere crypts or may be a half to three-quarters of an inch in length. They may open into the urethra just inside the meatus. Routh[1] believes them to be the efferent canals of Max Schüller's glands. Nagel, quoted by Clarence Webster,[2] thinks they are the para-urethral ducts, also described as the ducts of Malpighi. Others consider them to be the remains of the lower ends of Gartner's canals. Nagel denies this, maintaining that they are formed from depressions of the epithelium covering the uro-genital sinus.

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[1] Clarence Webster, *Diseases of Women*, 1898, p. 60.

[2] *Id. op. cit.* p. 61.

Specialised mucous glands and follicles have been described by Littré and Morgagni,[3] and are probably only particular foldings of the mucosa or more marked racemose mucous glands, as above described.

The interest of urethral glands arises in connection with urethral abscess and diverticula, to be described later.

Developmental Defects.—These have been partly referred to in malformations of the vulva, under epispadias and hypospadias, and in persistence of the uro-genital sinus. The urethra may rarely be completely wanting; if so it is incompatible with life unless a patent urachus exist at the same time.

Urethritis.—Inflammation of the urethra is frequently due to gonorrhœa[4] but it may follow the use of dirty instruments, or injury, or occur in connection with a new growth. The mucous membrane is acutely inflamed and there is either a catarrhal secretion or abundant purulent discharge. This can be squeezed out of the orifice by pressing from above downwards. Cystitis may co-exist, there may even be blood in the discharge, and the urethra may be felt as a thickened cord. The pus may contain gonococci. Skene's duct, may also be affected, and the vulvo-vaginal glands.[5]

Stricture of the Female Urethra.—This may occur, but it is rare;[6] it is discovered on attempting to pass a catheter. The female urethra will generally allow the passage of a No. 14 to No 17 male catheter. Strictures in the female may be due to congenital smallness of the urethra, injury during a difficult labour, or the whole urethra may be surrounded by thick fibrous tissue along its whole length. Herman thinks that this thickening of the urethro-vaginal cellular tissue is analogous to enlargement of the prostate in the male.[1]

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[1] A. Routh, 'Urethral Diverticula,' *Trans. Obstet. Soc. Lond.* vol. xxxii. 1890, p. 76.

[2] Clarence Webster, *op. cit.* p. 61.

[3] A. Routh, *op. cit.* p. 76.

[4] See chapter on 'Gonorrhœa in Relation to Pelvic Inflammation.'

[5] C. Webster, *op. cit.* p. 604.

[6] Herman, *Diseases of Women*, p. 648.

The most common cause of stricture in the young and middle-aged is gonorrhœa. It is very much rarer than in the male, but may sometimes occur. Other cases of stricture may be due to the fibrous overgrowths of esthiomène (lupus)[2] or from chancre or malignant disease.

Dislocation of the Urethra.—This may occur by its being pulled upwards by the uterus in labour, or from being dragged downwards by cystocele or prolapse of the womb. Large abdominal tumours, especially, fibroids may cause traction on the urethra. Retroversion of the gravid uterus acts much in the same way.

Chronic Congestion of the Urethra.—This is seen in pregnant women, or it may persist after delivery. Herman[3] refers to Sir C. M. Clarke and Dr. West's description. The urethra is swollen and tender, feeling like a cord as thick as the finger or thicker. Congestion may be accompanied by aching in the loins and frequent micturition.

Urethral Diverticula.[4]—In this condition there is a urinary pouch or cyst generally occupying the urethro-vaginal septum, which communicates with a urethra of normal calibre by a small orifice, usually in its middle third.

The **etiology** of such diverticula is much discussed. Routh[5] thinks they may arise from:

1. Closure of the ducts of pre-existing urethral glands, retention cysts resulting. Suppuration and ulceration into the urethra by a small, often valvular hole follows, and the inflammation is kept up by urine getting into the sac and decomposing. There is often congestion of the urethra. In such a case chronic abscess of the urethro-vaginal septum results,[6] lying between the urethra and the vagina forming a tense, convex, tender swelling. Sometimes there is no communication with the urethra; in others, the abscess from time

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[1] Herman, *op. cit.* p. 649.

[2] Herman, 'Stricture of the Urethra in Women,' *Trans. Obstet. Soc. Lond.* vol. xxix. 1887, p. 27; see also chapter on 'Diseases of the Vulva' in this book.

[3] Herman, *op. cit.* p. 661.

[4] A. Routh, 'Urethral Diverticula,' *Trans. Obstet. Soc. Lond.* vol. xxxii. 1890, p. 69.

[5] A. Routh, *op. cit.*

[6] Herman, *op. cit.* p. 662.

to time empties itself into that channel, filling again and again, and lasting for many years.

2. Blood cysts which have passed through similar changes.

3. The formation of pseudo-cysts by injury to the urethral floor during labour, or instrumentation.

The developmental origin of such diverticula is of interest. Routh thinks they may arise from Max Schüller's glands, and not from Skene's ducts. Gartner's ducts, which may be responsible for vaginal cysts[1] near the cervix, do not reach the urethra.

It is possible that dermoid tumours[2] may sometimes occur in connection with the urethra and discharge fatty matter.

It is a little difficult, in connection with these swellings, to find an adequate nomenclature, the word 'diverticulum' being usually employed to mean a congenital peculiarity. Possibly some of these abscesses opening into the urethra may be of this nature, but not all, as those due to injury during labour, with subsequent suppuration and blood extravasation, have a different origin. Some diverticula need not even start as cysts at all, but are akin to 'distension diverticula'[3] of the intestine or bladder, in which the mucous membrane protrudes through deficiencies in the muscular coat.

Tender Red Patches in the Urethra.—Herman[4] describes this condition and refers to Garstang's paper. It is marked by an extremely painful and vascular condition of the mucosa of the urethra, and there are patches on the wall, seen by the urethral speculum, of a bright red colour like caruncle. There is no tumour, but only these red patches or rings. It is not necessarily related to caruncle, and the spots are not erosions or ulcers. It causes severe pain on micturition or on passing a catheter.

Herman thinks that the condition, from its clinical appearance, is allied to the purple tender patches in Kraurosis vulvæ.[5] Associated with it there may be tender punctate vascular spots scattered over the vestibule.

Urethral Caruncle.—This should be described as a vascular tumour of the urethra, or angioma.[6]

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[1] Bland-Sutton, *Amer. Journ. Obstet.* vol. xiii.

[2] Herman, *Trans. Obstet. Soc. Lond.* vol. xxxii. 1890, p. 79.

[3] Doran, *Trans. Path. Soc. Lond.* xxix. p. 122.

[4] Herman, *Diseases of Women*, p. 665.

[5] Herman, *op. cit.* p. 667.

[6] See drawing, Howard Kelly, *Operative Gynæcology*, vol. i. pl. iv., opp. p. 307.

It is one of the commonest tumours of the urethra, and the most frequent cause of painful micturition in women.

The tumour consists of a bright red, smooth, vascular growth, generally springing from the lower border or posterior wall of the urethra. Sometimes the growth is rounded, at others flattened by lateral pressure of the labia, or it may be lobed like a cock's comb. It may or may not have a pedicle; some caruncles are sessile, others have a stalk reaching some way up the urethra. It is exquisitely sensitive. The tumour consists of connective tissue of a loose myxomatous nature, covered by layers of squamous or transitional epithelium investing hypertrophied papillæ. Caruncles are very vascular, and may bleed on examination. They contain blood spaces and cavities resembling those of the corpus spongiosum penis; a few well-formed vessels occur at the base, and the pedicle sometimes bleeds freely if the tumour be cut off with scissors, Bland-Sutton and Giles compare the blood spaces to the thin-walled vessels seen in piles.[1]

Herman, in investigating 41 cases,[2] found they were most common between the ages of forty and sixty. Kraurosis and red tender spots in the vestibule and around the hymen also occur with caruncle. A caruncle is rarely larger than a split pea, but some of the flat varieties may be of some size. No nerves have ever yet been actually demonstrated in microscopic sections.

Nothing is known as to their origin, although they are supposed to arise[3] from papillæ of congenital urethral cysts or from the erectile tissue of the urethra, or to changes taking place in Skene's ducts, possibly adenomatous.[4] It is significant that they are almost invariably situated on the posterior wall of the urethra. They are very troublesome to cure, and recur after removal, possibly, in most cases, because their base of attachment has not been thoroughly destroyed. They have no tendency to anything like malignancy.

Urethral Hæmorrhoids.—These are really varicose veins of the meatus, and appear as bluish-red swellings under the mucous membrane. They are generally quite small, sessile swellings bluish in colour like a pile, not bright red like a caruncle. They are not tender. They consist of dilated and thickened veins, and are unimportant.

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[1] Bland-Sutton and Giles, *Diseases of Women*, 2nd ed. p. 79.

[2] Herman, *op. cit.* p. 654.

[3] Englisch and Pozzi, quoted by Herman, p. 654.

[4] Bland-Sutton and Giles, *op. cit.* p. 79.

Prolapse of the Mucous Membrane of the Urethra.—This at first sight resembles caruncle. It occurs chiefly in children, and consists of descent of the mucous membrane all round the meatus, forming a protrusion 'like a purple frill,' having an opening in or near its centre.[1] It can be replaced if recent, but later, if neglected, it becomes œdematous, and can then only be reduced with great difficulty. It bleeds very readily, but is not sensitive like a caruncle. Herman records a rare case of Leech's in which the whole bladder protruded at the urethra, being turned inside out, forming a red tumour on which the openings of the ureters could be seen.[2]

Fibroma of the Urethra.—This appears to occur mostly in the urethro-vaginal septum, and appears as a firm, hard, well-defined tumour, sometimes bulging into the vagina. Such tumours, strictly speaking, do not belong to the urethra at all; they grow from the urethro-vaginal septum, and may be related to, if not identical with, vaginal fibroids. They may soften and undergo necrotic and gangrenous change if infected. Kelly[3] reports a case of this nature in which the tumour weighed nearly three pounds; it was an œdematous fibroid.

Sometimes tumours are seen growing from the urethra in young children,[4] generally quite small, and often pedunculated, which on section are composed of connective tissue. They may be included under the term fibroma. Myoma of the urethra is very rare; Kelly gives an account of one case.[5]

Cancer of the Urethra.—This is a very rare disease, and may affect the mucous membrane or the peri-urethral tissues, commencing in the urethro-vaginal septum.[6] Kelly[7] reports two cases of Reichel's, in which there was extensive carcinomatous affection of the entire urethra, which seemed to have taken its origin at the external orifice; the patients were both old women. Cases have been reported in which the growth protruded from the urethral orifice, or in which a nodule in the urethra had broken down, and a cancerous urethro-vaginal fistula resulted.

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[1] Herman, *op. cit.* p. 659.

[2] Herman, *op. cit.* p. 658.

[3] H. Kelly, *Operative Gynæcology*, vol. i. p. 310.

[4] H. Kelly, *op. cit.* vol. i. p. 309.

[5] H. Kelly, *ibid.* p. 311.

[6] Herman, *op. cit.* p. 660.

[7] H. Kelly, *ibid.* p. 311.

Kelly[1] relates two cases of peri-urethral cancer, in which the urethra was converted into a small rigid tube which bled easily and caused difficulty in micturition. Both cases were secondary: one to cancer of the labium, which had extended over the vestibule; the other to cancer of the cervix and vagina.

Sarcoma of the Urethra.[2]—This is a very rare disease. Kelly[3] reports four cases, including Galabin's. The latter occurred in a child of three years who had hæmaturia, and on examination a tumour three inches in diameter was found growing from the urethra, which was widely dilated; it had a red lobulated surface. It was a myxo-sarcoma. The other cases occurred in adults, and were lobed vascular tumours springing from the urethra, giving rise to bleeding and discharge.

Secondary sarcomatous nodules have been seen in the urethro-vaginal septum beneath the mucous membrane of the vagina.[4]

The author saw a case at the Samaritan Free Hospital, under Boulton, in which the disease was secondary to sarcoma (deciduoma) of the body of the uterus.

Tubercular Disease of the Urethra.[5]—This sometimes begins in the female urethra, and when present causes pain or incontinence of urine, accompanied by hæmaturia or pyuria.

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[1] Kelly, *op. cit.* p. 312.

[2] Galabin, *Trans. Obstet. Soc. Lond.* vol. xxxviii. 1896, p. 120.

[3] H. Kelly, *op. cit.* vol. i. p. 313, who quotes Ehrendorfer, *Centralb. f. Gyn.* 1892, No. 17, p. 321; C. A. L. Reed, *Amer. Journ. Obstet.* Dec. 1896, p. 864; Biegel, *Die Krankheiten des Weiblichen Geschlechtes*, vol. ii. Stuttgart, 1875, p. 654.

[4] Herman, *op. cit.* p. 660.

[5] Henry Morris, Allbutt and Playfair, *System of Gynæcology*, p. 928.

CHAPTER XVIII

DISEASES OF THE URETERS

Anatomy.[1]—The ureters are almost symmetrically placed, as flattish white cords in the loose connective tissue behind the peritoneum, extending from the pelvis of the kidney to the bladder. Each ureter is ten to twelve inches long, the left being longer than the right because of the higher position of the left kidney. The ureter begins at the pelvis of the kidney, and from thence its diameter is about three millimetres, but is a little narrower at each extremity. In the abdomen, the ureter leaves the kidney about one and a half inches from the median line, and curves down on the psoas muscle, at first a little inwards, then outwards, and crosses the brim of the pelvis one and a quarter inches from the middle line. Near its middle, the ureter on the psoas muscle is in relation with the ovarian veins and artery, and on the right side, above the brim of the pelvis, it lies behind the cæcum and the ascending colon. On the left it lies behind the sigmoid flexure at the brim, and above this behind the descending colon.

At the brim of the pelvis each ureter lies upon the common iliac artery, crossing it about one and a quarter inches from the middle of the sacral promontory; the ureter then descends, crossing the common iliac vein. The ovarian vessels cross the ureter and leave it at the brim, where they enter the top of the broad ligament.

In the pelvis the ureter runs at first behind the peritoneum close to the internal iliac artery, then it turns forwards, passing under the uterine artery, then beneath the broad ligament, passing through a sort of membranous foramen at its base (Kelly) midway between the cervix and pelvic wall. Beyond the cervix, it runs parallel to the upper wall of the vagina, which it crosses obliquely, forwards and inwards, entering the trigone of the bladder, which has already been described.

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[1] Howard Kelly, 'Affections of the Ureters,' *Operative Gynæcology*, vol. i. p. 396 *et scq.*

Howard Kelly[1] points out that the ureters are not mere passive tubes, but that the urine is passed down them by active peristalsis which expels a few drops of urine into the bladder every two or three seconds. Much has been done of late in ureteral surgery, and the subject is one of great importance.[2] The abdominal part of the ureter is perhaps not so important in diseases of women as the pelvic, and is not easily accessible to methods of examination.

The pelvic portion of the ureter can be palpated either by the vagina or rectum; by the vagina with the patient on her back in lithomy position, and the fingers passed high into the vaginal fornices, upwards and outwards, it is possible to feel the ureter as a flat cord which is constantly slipping away; it may be traced thus down the pelvic wall to a point at which it passes between the anterior vaginal wall and the bladder.[3] Sometimes the ureter is found lying close to the pelvic wall in the loose cellular tissue; if thickened by disease, as in tubercle, it feels like a hard cord. H. Kelly says that in advanced pregnancy, where the head is low in the pelvis, the ureters are markedly displaced and can be felt with extraordinary distinctness against the child's head. At its lower extremity the ureter may be confused with the obturator artery or nerve, or the tendinous arch of the levator ani. An inflamed ureter may be painful on touch, which may set up a desire to pass water. By the rectum, the ureter can be felt from the brim to the floor of the pelvis, especially on the left side, by passing the finger in the bowel, to near the bifurcation of the common iliac artery; then, passing it down behind and close to the internal iliac, a flat yielding cord can be traced downwards.[4] If a rubber bougie or catheter be first introduced into the ureter from the bladder, the course of the ureter is easily palpated. The methods of ureteral catheterisation[5] as described by Kelly should be consulted.

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[1] H. Kelly, *op. cit.* vol. i. p. 397.

[2] *Id. ibid.* p. 396 *et seq.*

[3] *Id. ibid.* p. 399.

[4] *Id. ibid.* p. 400.

[5] *Id. ibid.* pp. 400, 412.

CONGENITAL AFFECTIONS OF THE URETERS

Abnormalities of the ureters are not very common. They are most often found in non-viable fœtal monstrosities. Either one or other ureter may be absent or imperforate.

Double ureter is not uncommon, but has not any pathological significance. The ureters may be double on both sides, or on one side only, or they may start as a single cord, and then branch, or be double on leaving the kidney and fuse at some point below this. If the ureter is double below, it may have separate openings into the bladder.

Misplaced ureteral openings[1] occasionally occur in the vagina, or at the external urethral meatus (when a ridge can be felt in the vagina covering the ureter), or the ureter may open under the hood of the clitoris. In such cases there is persistent leakage of urine from birth, but at the same time the bladder acts normally and none of the ordinary vesico-vaginal fistulæ can be found. Howard Kelly describes the methods by which the condition may be diagnosed.[2]

Cystic Dilatation of an Occluded Ureter.[3]—This is very rare. The ureter fails to communicate either with the bladder or any part of the genital tract; in such cases curious cysts or sac-like dilatations may occur, and if low down they may project into the bladder, or form a bulging tumour into the vagina, imitating a vaginal cyst. Sometimes hydronephrosis has been observed, or the ureter may end blindly with no dilatation, the kidney on the same side being atrophic or congenitally malformed. Malformations of the uterus have also been observed with this condition. Congenital flexures or kinks of the ureter are sometimes met with.

Inflammation of the Ureter and surrounding Cellular Tissue.—Inflammation of the ureter may occur as an extension upwards from the bladder, as in septic infection or gonorrhœa, or from the kidneys downwards, as in tubercle, or it may arise from disease of the ureter itself, as in calculus.

Inflammation of the cellular tissue is generally due to infection extending upwards after operations on the bladder, or after vaginal hysterectomy.

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[1] H. Kelly, *Operative Gynæcology*, vol. i. p. 417.

[2] *Id. ibid.* pp. 418, 419.

[3] Orthmann, quoted by H. Kelly, p. 421; also Tangl, *Virchow's Arch.* vol. cviii. p. 414; Weigert, *Virchow's Arch.* No. 70, p. 490, quoted by H. Kelly, *op. cit.* vol. i. p. 422.

In most inflammatory conditions the ureter may be felt more or less distinctly, according to Kelly, as a thickened very tender cord in the lateral vaginal fornix, sweeping upwards to the vaginal vault and disappearing at the side of the cervix under the base of the broad ligament.[1] Sometimes it may feel nodular; by the rectum it may be still better distinguished.

In **Tubercular Inflammation of the Ureter**, most profound changes occur, and the ureter is affected throughout its entire length; it is generally secondary to a tubercular kidney (Kelly). The whole ureter becomes thick and rigid; ulceration and thickening of the mucous coat occur with much irregularity in the lumen of the ureter, or it may be practically obliterated.[2] The thickened rigid ureters can be easily felt by the vagina in such cases.

Obstruction of the Ureter.—This is of the greatest importance, owing to the serious effects upon the kidney, resulting primarily in dilatation of the ureter (Hydro-ureter), then of the pelvis of the kidney; these may at any time become infected and suppurate, giving rise to pyo-ureter and pyo-nephrosis, with hopeless destruction of the kidney, obstructive suppression of urine, and death.

Obstruction of the ureter is commoner in women than in men, and Kelly[3] gives an elaborate list of causes, according as the obstruction arises from without, or by foreign bodies in the canal, or by disease of the walls of the ureter itself.

Many large abdominal tumours or even fixed small tumours in the pelvis may cause obstruction, such as fibroids or ovarian tumours, cancer, inflammation of the cellular tissue through which the ureters pass, or tumours of the bladder itself.

A calculus, thick pus, or tubercular material, may block its lumen; chronic inflammation and cancer may affect the ureter itself. Some causes act on one side only, such as stone; some on both. Cancer of the cervix, spreading to the cellular tissue, is the most frequent cause of bilateral obstruction.

The amount of dilatation of the ureters in such cases is most remarkable.

The result of complete obstruction to the passage of urine is well known, but the symptoms may be delayed a week,[1] when twitching, stupor, and contracted pupils ensue; convulsions and coma sometimes occur, but are uncommon.

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[1] H. Kelly, *op. cit.* vol. i. p. 424.

[2] Bowlby, *Surgical Pathology*, 2nd ed. p. 414.

[3] H. Kelly, *op. cit.* vol. i. p. 431.

Stricture of the Ureter.[2]—Kelly gives a full account of cases and a valuable description of the methods of diagnosing the condition, and gives details of the way in which stricture may be detected, and the methods by which the ureter and even the kidney can be washed out. This should certainly be consulted.

The cause of such strictures may be due to gonorrhœa, tubercle, or injury. Sometimes a species of stricture results from kinks [3] in the ureters, which may be congenital, or due to inflammatory adhesions. The congenital ones, according to Kelly, are more likely to occur near the kidney; in such cases the obstruction may be intermitting, as evidenced by the kidney, which is in a state of hydronephrosis, emptying itself from time to time.

Calculus in the Ureter.—This is a much rarer condition than stone in the kidney or bladder. Calculi probably start in the kidney, and are forced down the ureter until they lodge somewhere in its course. Such calculi may be elongated; they gain by accretion at the ends, and lose by attrition at the sides. Kelly reports a calculus five inches long.[4] In the author's case at the Samaritan Free Hospital, in a woman of twenty-seven, the stone lodged about two inches from the bladder, and gave rise to intermitting attacks of pain and obstruction. It could be felt by the vagina close to the pelvic walls. As it appeared unwise to open the ureter in this situation, as subsequent sutures would have been impossible and the risks of extravasation very great, the stone was eventually removed by Henry Morris at the Middlesex Hospital by a sacro-coccygeal dissection. The stone measured about half an inch across in its greatest diameter, was cubical in shape, and formed of uric acid. The ureter was dilated where the stone existed, but was clear above and below this. The ureter was sutured and the wound drained.

Calculi in the ureter generally lodge just below the kidney, at the brim of the pelvis, or near the opening of the ureter into the bladder. Small stones may be passed into the bladder in the ordinary way, but large ones become fixed, and if they give rise to obstructive symptoms must be removed. In a case reported by Lane the stone had been in the ureter for twenty years.[1]

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[1] M. Duncan, *Clin. Lect.* 4th ed. p. 86.

[2] H. Kelly, *Operative Gynæcology*, vol. i. p. 431; *Johns Hopkins Gynæcological Reports*, No. 1; and *id. op. cit.* vol. i. pp. 438, 439.

[3] *Id. ibid.* p. 447; see fig. 250.

[4] *Id. ibid.* p. 449; fig. 252.

Prolapse of the Mucous Membrane of the Ureter into the Bladder.[2]—This is very rare; it is generally congenital, and occurs most frequently in female children. The prolapsed ureter may escape from the urethra and be mistaken for urethral prolapse. Kelly reports an acquired prolapse of the ureter in a man following on acute cystitis.[3]

Ureteral Fistulæ.—These may be traumatic, or due to ulceration from cancer, stone, or a pessary; they may also be congenital.

Injury to the ureter may occur from instruments in difficult labour, or by operations on the pelvic organs, such as vaginal hysterectomy. In the removal of large uterine fibroids there is a risk of injuring the ureter. Ureteral fistulæ nearly always occur in the lower portion of the ureter, near the base of the bladder, or the vaginal vault.[4] Kelly describes at length the various operations for the relief of this condition in the article on 'Affections of the Ureters' in his book on Operative Gynæcology.

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[1] Lane, *Lancet*, Nov. 8, 1890, p. 967. [2] H. Kelly, *op. cit.* vol. i. p. 455; fig. 257.

[3] Blumer, *Johns Hopkins Hospital Bulletin*, September 1896, p. 174.

[4] H. Kelly, *op. cit.* vol. i. pp. 457–477.

CHAPTER XIX

MICRO-ORGANISMS IN RELATION TO DISEASES OF WOMEN

It is not within the province of this work to enter into any detail on the subject of micro-organisms; but a short account of some of those principally concerned in gynæcological practice may not be out of place.

The subject is beset with much difficulty, and the whole of the genital tract must be considered, as a large number of its diseases result from infection due to microorganisms of one variety or another.

A very important problem is met with at the very beginning of this subject, viz.—Do micro-organisms inhabit the genital tract in health, or are they only to be considered as factors of disease?

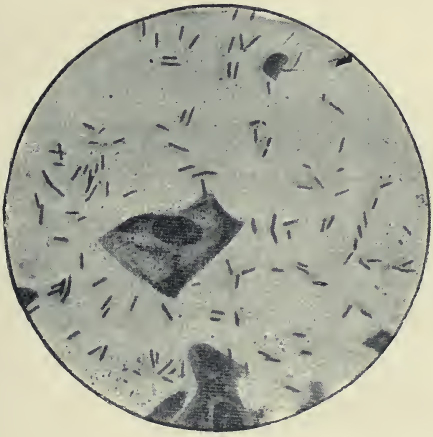
THE NORMAL SECRETIONS OF THE GENITAL TRACT

The normal secretions of the vulva are those of the sebaceous glands, sweat glands, and the glands of Bartholin. The urethra likewise contains many glands. The vulva is also moistened with the discharges which come from above, viz. those of the vagina and the uterus. That the vulval discharges must contain micro-organisms in the healthy woman is evident. The vulva is but a skin surface and exposed to the action of countless micro-organisms, both in health and disease, and almost the same remarks apply to the lower part of the vagina; hence the significance of the presence or absence of micro-organisms in a so-called normal discharge is often one of exceeding obscurity.

The normal secretions of the vagina have already been

Fig. 141.

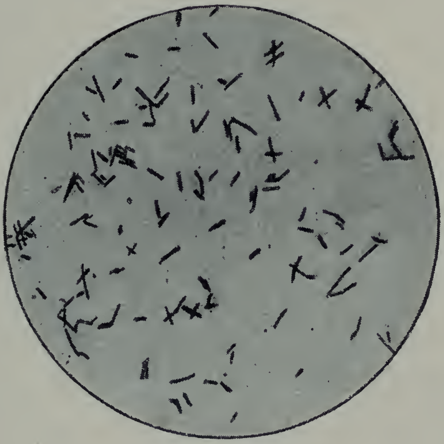
VAGINA BACILLUS.



Normal secretion from the vagina, showing the vagina bacillus. (Döderlein.)
(Bland-Sutton and Giles, 1st ed. p. 112.)

Fig. 142.

VAGINA BACILLUS.



Pure cultivation of the vagina bacillus. (Döderlein.)
(Bland-Sutton and Giles, 1st ed. p. 113.)

partly considered (p. 26). In health the vagina is certainly moist, and its secretion is thin, white, opalescent, and opaque, with a strong acid reaction. Gow has investigated this subject.[1] Döderlein[2] points out that even in health micro-organisms exist in the vagina. The latter has described a special *vagina bacillus* (figs. 141, 142), a rod-shaped organism belonging to the anaërobic bacilli,[3] on which the vaginal acidity in health depends. He believes that this bacillus has a protective or antagonistic action against pathogenic bacteria, and that if the latter are found in normal vaginal secretion they are always in a condition of weakened virulence.

If, however, the secretion of the vagina loses its acid reaction,[4] as, for example, during the period of the lochial discharge, the vagina bacillus disappears, and this safeguard against infection is overcome, its place being taken by ordinary pyogenic cocci. The vagina bacillus is present during pregnancy, and reappears under ordinary circumstances at the end of the puerperium. Krönig and others[5] point out that the normal secretions in the vagina contain a number of different bacteria, which can be cultivated under anaërobic conditions only. Krönig described two of these, both anaërobic non-pathogenic streptococci. It is claimed that but few aërobic or even facultative aërobic organisms are to be found in the vagina, and that the latter prefer acid media. Stroganoff[6] found that micro-organisms seemed to increase in abundance in the vaginal secretion preceding and following menstruation.

Whitridge-Williams, in examining the vaginal secretion in pregnant women,[7] agrees with Krönig that usually pyogenic cocci are absent, and that therefore 'auto-infection'[8] with them is impossible; and that when found in the puerperal uterus they have been introduced from without.[9] The same remarks apply to cases of death from puerperal infection. Whitridge-Williams rightly believes that all infection is conveyed by hands or instruments, and is due to neglect of aseptic precautions, and not merely to infection with organisms normally found in the vagina. Hence the value in obstetric practice of limiting vaginal examination and cultivating external palpation. More will be said of this later, but the rigid disinfection of anything which comes in *contact* with a puerperal woman cannot be too strongly insisted on, while vaginal douches, in ordinary cases, are unnecessary and harmful.

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[1] Gow, 'A Note on Vaginal Secretion,' *Trans. Obstet. Soc. Lond.* vol. xxxvi. 1894, p. 52.

[2] Döderlein, *Das Scheidensekret*, Leipzig, 1892.

[3] See p. 27. See also Reed, *Text-book of Gynæcology*, 1891, p. 164.

[4] See Howard Kelly, *Operative Gynæcology*, vol. i. p. 32; Bland-Sutton and Giles, *Diseases of Women*, 1st ed. p. 112.

[5] Howard Kelly, *op. cit.* p. 33.

[6] Quoted by Reed, *op. cit.* p. 165.

[7] J. Whitridge-Williams, *Trans. Amer. Gyn. Soc.* 1898.

[8] Auto-infection = infection with some organism which is a normal inhabitant of the vagina.

[9] Stroganoff finds that the vagina of the newborn child is free from micro-organisms, which, however, may enter soon after birth. See Reed, *op. cit.* p. 163.

Bacteria, therefore, appear to be constantly present in the vagina from the vestibule up to the cervix, and they may occur in the latter, in some cases, under normal conditions. It is evident, from what has already been said with regard to the bacteriology of the secretions of the external genital organs, that these may often contain a great variety, as well as a large number, of micro-organisms. Under such conditions the identification of such an organism as the gonococcus may present unsurmountable difficulties.

Of course, in cases of pelvic suppuration discharging into the genital tract, streptococci or staphylococci are generally found, either alone or in combination with other pathogenic micro-organisms.

The normal secretions of the uterus are those of the cervix and body. As pointed out before in this book, the cervical glands in health secrete clear, tenacious, glairy mucus, which is alkaline in reaction. Normally, the cervical canal is occupied by a plug of this material. The secretion of the body of the uterus is believed to be a thinner colourless mucoid fluid. During life little is known of it, as it is mixed with the secretions of the cervix. The occurrence of micro-organisms in the uterus is one of great importance. As a rule, from just within the os externum upward, the female genital tract in health is free from micro-organisms, while the vagina, as pointed out before, may contain all sorts of bacteria, the external os uteri being a sort of boundary line. This is an important fact in relation to pregnancy, for here the same holds good, that in health no micro-organisms exist in the uterus above the os externum.

With regard to the **Fallopian tubes in health,** all observers have come to the conclusion that they are free from bacteria. This is supported by the observations of Menge, Winter, and Sinclair.[1]

BACTERIA IN RELATION TO DISEASES OF THE GENITAL TRACT

With regard to the external genital organs, infection of the vulva, the glands of Bartholin, and the vagina, many of such diseases depend on the action of specific micro-organisms, and this is well shown by the liability of infection once established in the vulva or vagina to extend upward, and involve the endometrium, Fallopian tubes, and the peritoneal cavity; in fact, a proper comprehension of the general subject of infection of the female genitalia involves a study of the various pathogenic bacteria, a consideration of the micro-organisms known to be involved in the infection of these organs, and finally, a study of the infection, not alone of any one organ, but of the entire genital apparatus.[2]

Hence in speaking of diseases of women, such as vulvitis, vaginitis, endometritis, salpingitis, and the like, we may look on them as clinical entities; but from a bacteriological point of view such may be discussed as due to the action of micro-organisms of one variety or another.

With regard to the **vulva,** almost any variety of micro-organisms may be found, particularly the gonococcus, streptococci and staphylococci, and the tubercle bacillus.

The special bacteriology of soft sore and syphilis will be described later, and it is more than probable that many of the so-called skin affections of the vulva are dependent on the action of micro-organisms.

Practically the only organism of importance found in the **urethra** in disease is the gonococcus.

With regard to the **vagina,** as pointed out by Döderlein and Whitridge-Williams, the normal acid secretion should only contain the so-called vagina bacillus, but pathological discharges may contain pus corpuscles and all kinds of bacilli, such discharges often having a neutral or alkaline reaction; the organisms most frequently met with being gonococci, streptococci, and staphylococci. The tubercle bacillus and gas-producing organisms [1] are very rare.

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[1] Reed, *op. cit.* p. 484–487.

[2] Reed, *op. cit.* p. 163.

Practically, infection of the vagina comes from without; the vagina allows the entrance of any micro-organism, as in a way, like the vulva, it is exposed to the air. Again, organisms may be introduced by digital examinations, the use of dirty instruments, and so on.

Nothing is more unsatisfactory in many cases than the examination of vaginal discharges from a bacteriological point of view. A patient has a discharge, and some of this is collected on a swab of wool and sent up to the laboratory for a bacteriological report, possibly with the idea of determining whether the discharge is gonorrhœal or not. Examination of such material in most cases proves little or nothing. The discharge swarms with micro-organisms of all kinds, pathogenic and non-pathogenic, and it is impossible to give an opinion other than 'gonococci have or have not been found.'

The interest of the micro-organisms of the vulva and vagina lies in the possibility of their transference to the higher parts of the genital tract, the uterus, tubes, and peritoneum. But it is possible that the existence of large numbers of pathogenic bacteria in the vagina is compatible with health; in fact such must be the case, but if these be transferred to the uterus serious results may ensue.

This is of great importance in relation to asepsis and antisepsis in obstetric practice, and to the means by which infection can be prevented or diminished. Whitridge-Williams [2] finds that the vaginal secretion of pregnant women does not contain the ordinary pyogenic cocci, having found the staphylococcus epidermis albus only twice in ninety-two cases, but never the streptococcus pyogenes, or the staphylococcus pyogenes aureus or albus, and he further does not believe that 'auto-infection' in puerperal fever is possible.

The observations of Pfannenstiel,[3] Döderlein,[4] Gönner,[5] and others, on the lochia in the puerperium, show the importance of the streptococcus in puerperal sepsis.[1]

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[1] Herman, *Diseases of Women*, p. 441.

[2] Whitridge-Williams, *Trans. Amer. Gyn. Soc.* 1898, and Reed, *op. cit.* p. 165.

[3] Pfannenstiel, 'Kasuistische Beiträge zur Aetiologie des Puerperalfiebers,' *Centralb. f. Gyn.* 1888, p. 617.

[4] Döderlein 'Micro-organisms in the Lochia,' *ibid.* 1888, No. 23.

[5] Gönner, 'Ueber Micro-organismen im Secret der Weiblichen Genitalien während der Schwangerschaft und bei puerperalen Erkrankungen,' *ibid.* 1887, p. 444.

The gonococcus is occasionally found in the vaginal secretion, and during the puerperium may extend from the cervix into the uterus and tubes.

It is perhaps out of place in such a work to speak of 'meddlesome midwifery,' but incalculable harm is done by frequent vaginal examinations and vaginal douching, these being the very means by which septic organisms may be introduced.

Bacteria found in the uterus are introduced from without. This has already been pointed out, for in health none exist above the os externum.

Winter found that the healthy uterine cavity contains no micro-organisms ; but that the cervical secretion might contain numerous bacteria, and in pregnancy the bacteria—especially the bacilli—increase to a large extent. These statements coincide with other German bacteriologists, including Lomer and Bumm.[2] Gönner, who made numerous observations, found bacteria in the cervix of pregnant women, but he failed to cultivate any. Menge,[3] however, finds that, with the exception of the gonococcus, no bacteria are found as a rule in the cervix of pregnant women.

Stroganoff made observations on women during menstruation. After complete cleansing of the external os, the canal of the cervix was found free of bacteria. In elderly women he found the cervical canal free from bacteria in 50 per cent. of cases. When the uterus was prolapsed, bacteria were found in small quantities in the cervical canal.

In pregnant women under ordinary conditions, he always found the canal free from bacteria. Stroganoff therefore concluded that in normal circumstances the cervix does not contain bacteria, the normal cervical secretion possesses a bactericidal quality, and in the genital canal the os externum forms the dividing line between the germ-containing and the germ-free portions.[4]

Turning now to **bacterial infection of the endometrium**, the subject becomes one of great importance, not only in diseases of women, but in obstetrics, for in the latter it underlies the whole subject of so-called puerperal fever; while in the former, infection of the uterus is but a stage of the spread of infection to the tubes, ovaries, and peritoneum. The subject is a vast one, and comprises many of the diseases called 'endometritis,' and, as pointed out by Barbour,[1] it has been suggested that a classification may be made of endometritis in two great groups, according to whether they are due to micro-organisms or not. Winckel classifies those due to micro-organisms as gonorrhœal, tubercular, puerperal (septic), saprophytic (of which senile purulent endometritis is probably one form), the so-called diphtheritic endometritis (which is really due to streptococci), syphilitic endometritis, endometritis due to fungi, and endometritis due to amœbæ.

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[1] A. H. F. Barbour, article 'Inflammation of the Uterus,' Allbutt and Playfair, *System of Gynæcology*, p. 207.

[2] Reed, *op. cit.* p. 353.

[3] *Ibid.* p. 354.

[4] *Ibid.* p. 353.

In endometritis much of the infection is necessarily of a mixed type, and it is possible that many organisms play their part, though such organisms as the streptococcus pyogenes, the gonococcus, and the tubercle bacillus may exercise a predominating influence in producing pathological changes, and these changes may not be limited to the endometrium itself, but spread to the deeper tissues of the uterus, so that endometritis and metritis co-exist. It is particularly with puerperal infection, or with a uterus containing a gangrenous fibroid, that changes are found to which it seems justifiable to give the name 'metritis,' and acute endometritis is probably always associated with a certain amount of metritis. Whether micro-organisms play such an important part in chronic endometritis is doubtful; but it is possible that, although no organisms can be detected, they have been present and have disappeared.[2]

The causes and pathological changes in endometritis have already been considered in Chapter VI. New growths of the uterus, such as fibroids and cancer, may at any time undergo necrotic changes if infected from without by micro-organisms. A good example of this is seen in fig. 67, while similar remarks apply to the infection of retained clots and products of conception in obstetric practice, the dangers and results of which are but too well known.

Extension of gonorrhœa to the endometrium results generally from mechanical intervention. It rarely occurs spontaneously. As pointed out before, although the cervix may be affected in a large number of cases, it is rare to find gonococci in the body of the uterus. In Schultze's cases,[1] where the cervix was affected with gonorrhœa, gonococci were found in the uterus in only 38 per cent., while in those cases where the uterus was affected the tubes and ovaries were also infected in 45 per cent. of the cases.

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[1] A. H. F. Barbour, *op. cit.* p. 206.

[2] Reed, *op. cit.* p. 359.

Tubercular infection of the endometrium has already been considered, but it is curious that the body is affected in a very much larger number of cases than the cervix. Tuberculosis of the body of the uterus is common in cases of general tuberculosis; in fact, after the Fallopian tubes, the endometrium stands next in order of infection by tubercle.

Nothing definite is known of syphilitic infection of the endometrium, though the tendency of syphilitic women to miscarry is frequently put down to this cause.

The Bacteria of the Fallopian Tubes in Disease.—The importance of diseases of the Fallopian tubes in relation to pelvic inflammation has already been pointed out in Chapter III. Still more interesting are the micro-organisms found under these conditions in the tubes themselves. Salpingitis and its results are considered in Chapter X.

So far as is known, no micro-organisms have been discovered in ordinary hydrosalpinx, but in pyosalpinx and purulent salpingitis the gonococcus is by far the most frequent organism that is found, though other organisms occasionally occur, such as streptococci, staphylococci, and the tubercle bacillus. The bacillus coli communis has been found in cases where the diseased tube is adherent to adjacent viscera. In the vast majority of cases the infection of the Fallopian tubes is the result of extension from below, endometritis preceding the salpingitis.

Much interest attaches to the tubercle bacillus in relation to diseases of the tubes, and as to whether the disease is primary or secondary in the majority of cases. This has already been treated of in Chapter X. Blood infection is also an important point. Cases of pyosalpinx occur where the pus on examination is quite sterile, the bacteria which caused it having died out. This is particularly so with the gonococcus. Careful examination of such tubes should be undertaken, for, as Targett[1] pointed out, unless careful sections are made of the uterine end their tubercular nature remains unsuspected. It is impossible to tell at an operation whether a pyosalpinx is tubercular or not. It is probable that in many chronic cases of pyosalpinx the pus is quite sterile, which may account for the absence of temperature in such cases and for the comparative safety with which such tubes can be removed by operation, even if they are burst during the process.

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[1] Reed, *op. cit.* p. 372.

Probably the same micro-organisms which give rise to pyosalpinx are instrumental in the formation of abscess of the ovary. This is particularly the case with gonorrhœa.

Streptococcus infection of the tubes is but a part of the general septic processes which may follow labour, abortion, or operations on the uterus. In such cases it is, of course, possible that the infection may not extend beyond the tubes, the inflammation being limited by perimetric closure of the fimbriated end, though in cases of rapidly fatal sepsis this unfortunately does not occur, and the general peritoneal cavity is involved.

With regard to infection of the **broad ligaments and pelvic peritoneum,** various bacteria may be found. They include the streptococci, staphylococci, and the tubercle bacillus. They are practically identical (except the gonococcus) with those found in the endometrium and Fallopian tubes, and as a rule result from infection of the genital tract from below. As already pointed out in the chapter on Pelvic Inflammation, in pelvic peritonitis the infection reaches the peritoneum through the uterus and Fallopian tubes; while in pelvic cellulitis the extension is practically a direct lymphatic one, often from some lesion of the uterine tissues, which opens up the cellular spaces. In puerperal sepsis both the peritonitis and the cellulitis may be evidences of streptococcus infection.

The gonococcus possibly plays some part in pelvic peritonitis, or, at all events, in those diseases of the Fallopian tubes with which peritonitis is associated, such peritonitis, in fact, being a conservative process which limits the spread of further infection to the general peritoneal cavity. Probably gonococcus infection in pelvic cellulitis is very rare.

Tuberculosis of the peritoneum is, of course, not a very uncommon affection, the broad ligaments being affected in the course of the disease. In all cases it is due to the invasion of the peritoneal cavity by the tubercle bacillus, but the method of invasion varies in different cases. In a large number of cases the disease is primary; but Whitridge-Williams brings forward evidence that infection may occur through the genital tract, which may account for the greater frequency of tubercular peritonitis in women than in men.[1]

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[1] Targett, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 163.

The part that the bacillus coli communis plays in peritonitis is doubtful, but some cases of infection and suppuration of ovarian cysts or fibroids (which are already in a state of low nutrition) seem to be often due to this micro-organism alone. This may also occur in ovarian cysts with a twisted pedicle, especially if associated with adhesions to neighbouring intestine.

Infections of the ovary itself, apart from ovarian tumours, may occur in the course of a general septic infection of the genital tract; this is seen in sloughing ovaries in puerperal cases, or with the multiple abscess formation due to streptococcic or gonorrhœal infection. Sometimes a definite ovarian abscess results, and in nearly all cases the ovary and tube are very closely adherent. In such cases infection by the bacillus coli communis is always possible. Tubercle of the ovaries may occur as a part of general tuberculosis or tubercular peritonitis.

The bacteria found in the **urinary tract** in the female are those in connection with urethritis, cystitis, and the suppurative conditions of the ureters and kidneys. Attention has already been drawn to this point in Chapters XVI., XVII., and XVIII. Many varieties of bacteria have been found in the bladder.[2] The ordinary pyogenic organisms, streptococci and staphylococci, reach the bladder from without, generally by dirty catheters, from an abscess opening into the bladder, or in connection with foreign bodies or new growths of the bladder itself. The tubercle bacillus is a common cause of chronic cystitis, and usually infects the bladder from a focus in the kidneys.

The gonoccocus almost always enters the bladder through the urethra, and is said to be a cause of cystitis in some cases, especially those occurring after labour.

[1] Reed, *op. cit.* p. 692.

[2] Reed, *op. cit.* p. 792.

The bacillus coli communis is frequently present in the bladder. It probably reaches the bladder either from the kidneys or through the urethra, or passes directly from the bowel to the bladder when these organs are connected by inflammatory adhesions. Neither the colon bacillus nor the gonococcus decomposes urea, and the urine remains acid in these infections.

A large number of non-pathogenic organisms are capable of producing alkaline fermentation in urine.

PATHOGENIC MICRO-ORGANISMS MET WITH IN GYNÆCOLOGICAL PRACTICE.[1] DESCRIPTION OF SPECIAL VARIETIES.

Having shortly described the particular portions of the genital canal where various micro-organisms in relation to this subject are to be found, the author proposes to give a short account of some of the varieties of special interest. For further information works on Bacteriology must be consulted.[2]

The Bacteria of Suppuration.—A considerable number of bacteria have been described as occurring in acute suppuration, some have been proved capable of producing the condition, but in others the pyogenic power has not been fully determined.

The streptococcus pyogenes, the staphylococcus pyogenes aureus, staphylococcus pyogenes albus, and staphylococcus pyogenes citreus are those generally included under the term micro-organisms of suppuration.[3] The gonococcus is a pyogenic organism, and the bacillus coli communis and the pneumo-coccus are commonly found, while others [4] occur less frequently, but the pus-cocci first mentioned are those most usually met with. Suppuration may also be produced by the tubercle bacillus.

Staphylococcus pyogenes aureus. (Fig. 143.)—This is a spherical coccus, which generally grows in clusters or masses. It stains easily with the ordinary aniline dyes, and it retains

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[1] Howard Kelly, *Operative Gynæcology*, vol. i. p. 35. Drummond Robinson, 'On certain Micro-organisms of Obstetrical and Gynæcological Interest,' *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 263.

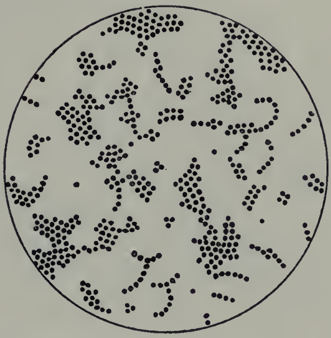
[2] Sternberg, Muir and Ritchie, etc.

[3] Howard Kelly, *op. cit.* p. 39.

[4] 'Micrococcus pyogenes tenuis, staphylococcus cereus albus, staphylococcus cereus flavus, bacillus pyocyaneus, micrococcus tetragonus, pneumobacillus, and others.' Muir and Ritchie, *Manual of Bacteriology*, 2nd ed. p. 166.

Fig. 143.

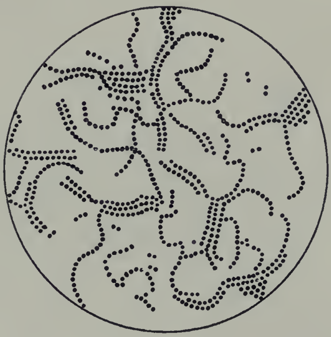
STAPHYLOCOCCUS PYOGENES AUREUS.
(Culture.)



Composite diagram.

Fig. 144.

STREPTOCOCCUS PYOGENES.



Semi-composite diagram of pure culture of Streptococcus pyogenes. (High power.)

the colour in staining by Gram's method. It grows readily on all ordinary media, and readily liquefies gelatine, the growth which falls to the bottom assuming a bright orange colour; hence its name. It is to be found in the air and dust, and on almost all cutaneous and mucous surfaces of the human body. Outside the body it has great tenacity of life. Sternberg[1] gives the thermal death-point in fluid media as from 56° to 58° C., with a ten minutes' exposure, though this is probably too low;[2] but when dried as 90° to 100° C.

Staphylococcus pyogenes albus is a similar organism in its morphological and biological characters and in its staining reactions, but the growth on all media is white.

The staphylococcus pyogenes citreus is less frequently met with. It differs from the staphylococcus pyogenes aureus and albus in the colour of the cultures, which are a lemon yellow, and is stated to be less virulent than the other two.[3] The staphylococcus cereus albus and staphylococcus cereus flavus are less important.

Staphylococci are found in many inflammatory processes, either alone or in combination with other organisms. They are found commonly in inflammatory processes which tend to remain local, *e.g.* boils and localised abscesses, and are often present alone in infective endocarditis and pyæmia. The staphylococcus pyogenes aureus is the most virulent, the staphylococcus pyogenes albus next, but the virulence of all of them is variable.[4]

Streptococcus pyogenes. (Fig. 144.) — This grows in chains, which may be of considerable length, especially when growing in fluids, the size of the individual cocci in older cultures being variable. They stain readily by Gram's method and with ordinary aniline dyes. The streptococcus pyogenes grows more slowly in cultures than the staphylococcus; it is less resistant outside the body, and dies out more readily[5] in culture. It does not liquefy gelatine.

The streptococcus pyogenes, as compared with the staphylococcus pyogenes aureus, is found more commonly in spreading inflammatory processes, *e.g.* erysipelas, &c., but is found in many acute abscesses and in infective endocarditis. It may cause local suppuration in any part of the genital tract, and may invade the lymphatics or blood-vessels, causing peritonitis or even septicæmia.

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[1] Sternberg, *Manual of Bacteriology*, 1893 p. 267; Muir and Ritchie, *op. cit.* 2nd ed. p. 168.

[2] It is probably about 70° C.

[3] Muir and Ritchie, *op. cit.* 2nd ed. p. 168.

[4] Howard Kelly, *op. cit.* p. 40.

[5] Muir and Ritchie, *op. cit.* 2nd ed. p. 169.

Most cases of puerperal fever [1] and puerperal peritonitis are due to this organism, in which condition it may be found after death in the various organs, though examination of the blood during life may give negative results. In pyæmia it is frequently present, though not always in pure culture.

It is often present as a mixed infection in tuberculosis. Probably the streptococcus pyogenes and the streptococcus of erysipelas are identical.

Secondary abscesses in lymphatic glands are more frequently caused by streptococci than by staphylococci.[2]

The Bacillus coli communis. (Fig. 145.)—This organism grows in the form of short thick rods with rounded ends, occasionally forming long threads. It stains with ordinary aniline dyes, but does not stain by Gram's method. It does not form spores. Gas-formation is easily demonstrated in appropriate cultures. This bacillus is the chief organism present in the large intestine in normal conditions; it also inhabits the small intestine. It resembles very closely the typhoid bacillus.[3]

The bacillus coli communis is found in a great many inflammatory and insuppurative conditions associated with lesions of the intestine, and suppuration in the peritoneal cavity. It is frequently present in inflammation of the urinary passages, such as cystitis, pyelitis, and abscess of the kidney. Cultivations of this bacillus from various lesions are more virulent than those obtained from the normal intestinal contents.

Reed points out that it is a well-established cause of ovarian abscess in a small percentage of cases, but that a colon bacillus infection never occurs except in an ovary which has previously been adherent to the bowel.[4] Suppuration of ovarian cysts, especially after twisting of the pedicle with resulting adhesion to bowel, may be explained in the same way.

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[1] Muir and Ritchie, *op. cit.* 2nd ed. p. 179. See also Reed, *op. cit.* p. 52, who quotes Czerniewski's observations on the lochia of women with puerperal fever, in which it occurred in 35 out of 38 cases, and in ten fatal cases it was present in the lochia before, and in the organs after, death

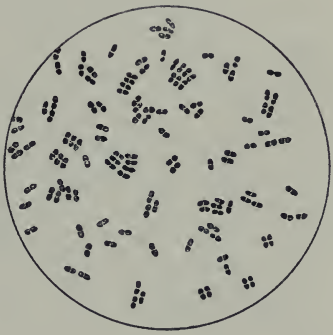
[2] Muir and Ritchie, *op. cit.* 2nd ed. p. 178.

[3] *Ibid.* pp. 324–330.

[4] Reed, *op. cit.* p. 575.

FIG. 145.

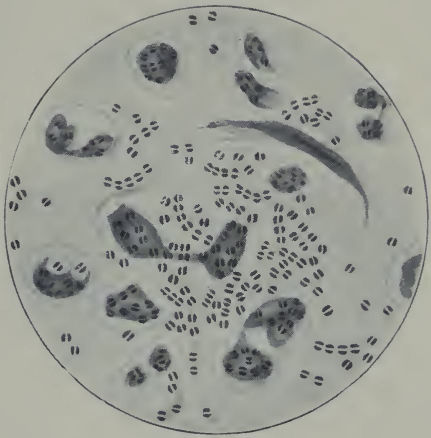
BACILLUS COLI COMMUNIS.



From a culture.

Fig. 146.

GONOCOCCI.



Composite diagram of gonococci in pus, to show their shape and the arrangement in pairs. Many are actually contained within the nuclei of the cells, which they appear to have partially destroyed. (High power.) Modified from Howard Kelly.

The Gonococcus. (Fig. 146.)—Reference has already been made to this in Chapter IV., and only its special characters will be described here. The gonococcus of Neisser has been shown conclusively to be the cause of gonorrhœa. It occurs in pairs or in groups of four, but generally in the form of diplococci, the adjacent margins of the two cocci being flattened or even slightly concave, so that between them there is a small oval interval that does not stain.

They may be likened to two beans placed side by side.[1] Sternberg uses the term biscuit-shaped,[2] in describing their appearance. Irregular degenerate forms often occur. The gonococcus stains readily with the ordinary aniline dyes, but becomes decolorised when Gram's method is employed. It practically will not grow on the ordinary culture media, and even on special media[3] the growth soon reaches a maximum, and the organism dies out if not subcultured at short intervals. In cultures, degeneration forms make their appearance very early.

The gonococcus is found in large numbers in the pus of acute gonorrhœa, characteristically contained within the leucocytes, though a few are seen, especially in the earliest stages, lying outside the leucocytes. In more chronic cases the number of the organisms becomes much reduced, and other organisms are then commonly present. Gonococci are found as the cause of gonorrhœa in the mucous membrane of the urethra, the vulvo-vaginal glands,[4] and the cervix uteri. In the opinion of the author, gonococci do not infect the vagina of the adult (though they may be present there), but they may do so in the gonorrhœal vulvo-vaginitis of young children. Infection of the lower part of the genital canal in women may spread to the endometrium,[5] Fallopian tubes, and the pelvic peritoneum, while from the urethra the infection may involve the bladder and even the kidneys; but the infection in cystitis and pyelitis is not usually pure.

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[1] Muir and Ritchie, *op. cit.* 2nd ed. p. 189.

[2] Sternberg, *op. cit.* 1893, p. 283.

[3] Wertheim's is the most used (human blood serum one part, agar two parts).

[4] Reed, *op. cit.* pp. 244–245.

[5] Schultze has proved that infection of the body of the uterus by gonococci is uncommon, while the cervix is very frequently involved. See Reed, *op. cit.* p. 372. See also A. H. F. Barbour's article, 'Inflammation of the Uterus,' Allbutt and Playfair, *System of Gynæcology*, p. 206, and Reed, *op. cit.* p. 352 *et seq.*, 'The Bacteria of the Uterus.'

As pointed out by Krönig, gonorrhœal infection of the uterus in the puerperium occurs in some cases, with high temperature and a purulent discharge. The occurrence of purulent ophthalmia in the child and the characteristic micro-organisms in the lochia would confirm such a diagnosis. In some such cases very severe septic symptoms have resulted, combined with arthritis and endocarditis,[1] but it is probable in some of these that the gonococcus infection is associated with a secondary one due to the streptococcus pyogenes.

During the past few years the presence of gonococci in the pus of certain cases of pyosalpinx[2] has been demonstrated. The cultures have generally been pure, but occasionally other bacteria have been found.

Wertheim[3] produced a typical gonorrhœa with cultures from a pyosalpinx. Gonococci have been detected in the pus found in the pelvis of a patient on whom abdominal section had been performed for suppurating tubes; the pus which escaped from the tubes having caused peritonitis.[4] It may be stated as a broad principle that the gonococcus is by far the most frequent cause of purulent salpingitis.

Sometimes the pus of a pyosalpinx is sterile, but in such cases it is possible that gonococci, or other organisms, have been present, but have subsequently died out. Gonococci have been found on the surface of the ovary and in the pus of ovarian abscesses.

The implication of joints probably takes place by the blood-stream; the micro-organisms cannot be found in all cases, but in some they certainly occur, and pure cultures have been obtained.[5]

With regard to the diagnostic value of microscopical examination alone in gonorrhœa, the presence of a large number of micro-cocci in a urethral discharge, having certain characters, position (intra-cellular), and staining reactions, is practically conclusive; but when few gonococci are present, and especially when they are mixed with other organisms, it is impossible to give a definite opinion. In such cases, plate cultures could alone supply the absolute test,[1] though the number of other organisms present is often so large as to render even this method of no avail.

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[1] Osler, *Principles and Practice of Medicine*, 3rd ed. p. 256, and Thayer and Lazear, *Journal of Experimental Sciences*, vol. iv. No. 1, 1899; *Johns Hopkins Hosp. Bulletin*, 1896, vii. p. 57.

[2] Drummond Robinson, *op. cit.* p. 278.

[3] Wertheim, *Arch. f. Gyn.* vol. xlii. 1892, and *Centralb. f. Gyn.* vol. xvi. 1892, p. 385.

[4] Drummond Robinson, *op. cit.* p. 278; see also Cullingworth, 'Etiological Importance of Gonorrhœa,' *Brit. Med. Journ.* June 20, 1899; Lewers, *Trans. Obstet. Soc. Lond.* vol. xxvii. 1885, p. 298.

[5] Muir and Ritchie, *op. cit.* 2nd ed. p. 197, who quote Neisser, Lang, Bordoni-Uffreduzzi, and others.

Soft Sore.—Ducrey and Unna[2] have described a special bacillus in connection with this condition. It occurs in the form of minute oval rods, forming groups or short chains, especially in the deeper tissues. The bacillus is said to be always present in the discharge from the sore. Unna detected it in the infected tissues. It has hitherto been observed in no other form of ulceration, and is sharply marked off from the ordinary saprophytic organisms by the fact that it has not been obtained in cultures outside the body.[3] It is possible that buboes associated with soft sore are caused by the same micro-organism. Absolute proof is wanting, as pure cultures have not been obtained.

Syphilis.—Regarding the relation of bacteria to this disease little is at present known, though Lustgarten has described an organism resembling the tubercle bacillus found in the primary sore and in the lesions in internal organs.[4]

The organism has not been cultivated outside the body, and it has been found in the tertiary lesions, which are generally supposed to be non-infectious. Most observers think the relations of Lustgarten's bacillus to syphilis are still highly problematical.

The Smegma Bacillus.—This is of some importance as it somewhat resembles the tubercle bacillus in form and staining reactions, though it is rather shorter and shows other minor morphological differences.[5] It occurs in the smegma of the external genitals, and may be found, especially in women, in the urine, into which the smegma gets detached. It can be readily decolorised, by alcohol, after the usual treatment with mineral acids, by which means it may with care be distinguished from the tubercle bacillus. This organism is not pathogenic, and in any doubtful case inoculation experiments would settle the matter.

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[1] Muir and Ritchie, *op. cit.* 2nd ed. p. 199.

[2] *Ibid.* p. 199; see also Reed, *op. cit.* p. 183.

[3] *Ibid.* p. 201, and Reed, *op. cit.* p. 186.

[4] *Ibid.* p. 202.

[5] *Ibid.* p. 256, fig. 68.

Tubercle. (Fig. 147.)[1]—It is unnecessary to give a detailed account of the tubercle bacillus in a work on gynæcological pathology, though its importance in many of the diseases of women cannot be passed over without mention. Reference has already been made to this in the various chapters on diseases of the ovaries, uterus, Fallopian tubes, &c.

The portal of entry into the genito-urinary tract is not always apparent.[2]

Tubercle bacilli (fig. 146) occur as minute rods, which usually measure $2{\cdot}5\mu$ to $3{\cdot}5\mu$ in length, and ${\cdot}3\mu$ in thickness; *i.e.*, in proportion to their length, they are comparatively thin organisms.

They are straight or slightly curved, and fairly uniform in thickness, but may show slight swelling at their ends. It has not yet been definitely proved that spore-formation occurs.[3]

They stain[4] with great difficulty, but once stained they resist the decolorising action of mineral acids in common with certain other organisms belonging to the 'acid-fast' group.

Tubercle bacilli retain their vitality for a considerable period outside the body, resembling in this respect bacilli which contain spores. They also resist drying and putrefaction, but are rapidly killed by exposure to direct sunlight.

The lesion caused by tubercle is the well-known nodule consisting of a central giant cell, surrounded by epithelioid cells, with an outer zone of small-celled infiltration, but the structure often varies.

In tubercular lesions bacilli may be easily found, especially in acute cases, or when caseation is rapidly spreading. In chronic cases they may be few in number and difficult to detect.

They occur in the discharges from tubercular lesions, such as the sputum, fæces, or urine. Infection in all cases probably occurs by the inhalation or ingestion of tubercle bacilli, which may then eventually involve internal organs. Local infection

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[1] See Osler, *op. cit.* 1892, p. 184.

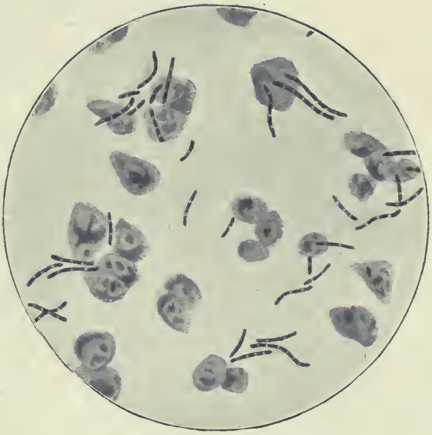
[2] Howard Kelly, *op. cit.* vol. i. p. 38. See, however, Reed, *op. cit.* p. 176.

[3] Muir and Ritchie, *op. cit.* 2nd ed. p. 228. To this excellent account of tuberculosis, the reader is referred for further details.

[4] *Ibid.* p. 113; see also Howard Kelly, *op. cit.* vol. i. p. 38–39.

Fig. 147.

TUBERCLE BACILLI.



Composite drawing. (High power.) Modified from Howard Kelly.

of the genito-urinary apparatus is possible, but it has not been satisfactorily proved.

The various diseases of women due to tubercular infection have already been described in former chapters of this work, and will here be only shortly referred to, though they are of extreme importance.

Tubercle of the vulva and vagina is very rare. Howard Kelly figures a case that occurred in the vestibule.[1] Tuberculosis of the vagina is usually associated with tubercle of the higher parts of the genital tract, probably infected from the discharges from above. Friedlander reports a case where a vaginal ulcer was the only tuberculous lesion that could be discovered in the entire body.[2] Reed thinks that the infrequency of the disease in both the vagina and vulva as compared with that of the uterus, and tubes, &c., is to be explained by the resistance of squamous epithelium to bacterial invasion. A few cases of tuberculous vaginitis that have been reported consisted in an eruption of miliary tubercles either in the posterior fornix or over the entire vaginal wall. These subsequently broke down into ulcers, which had a serpiginous shape, covered with caseous material.[3] Further ulceration may lead to fistulæ either into the bladder or bowel. Tubercular lesions of the vulva and vagina generally occur during the active period of sexual life.

Tuberculosis of the uterus[4] is generally associated with tubercular lesions elsewhere, such as the Fallopian tubes, peritoneum, and lungs. It probably occurs oftener than is thought, as careful post-mortem examinations of patients dying of phthisis have proved.[5]

Tubercle may occur in the cervix, but it is a rare condition. The ulceration in such cases resembles malignant disease, till subjected to microscopic examination.

Tubercle of the body of the uterus affects the endometrium,[6] and may be a primary or a secondary condition, and is frequently associated with tubercular disease of the Fallopian tubes.

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[1] Howard Kelly, *op. cit.* vol. i. p. 200.

[2] Reed, *op. cit.* p. 175.

[3] Reed, *op. cit.* p. 176.

[4] Herman, *op. cit.* p. 40; Reed, *op. cit.* p. 384.

[5] Turner, *Trans. Obstet. Soc. Lond.* vol. xli. 1899, p. 161, plate 4.

[6] Cullen, *Johns Hopkins Hospital Reports*, vol. iv. p. 91; H. Kelly, *op. cit.* vol. i. p. 489, plate 8; Reed, *op. cit.* p. 388, who thinks that the endometrium may be infected from above or below.

Pathologically, the endometrium may be found studded with miliary tubercles, or the whole uterine cavity is found ulcerated and filled with cheesy pus. Sometimes the ulceration may reach the muscular layers, which are found infiltrated, and later become destroyed.

A mixed infection of pyogenic cocci and tubercle bacilli, associated with some obstruction at the os internum, will lead to pyometra.[1] Sometimes the tubercular process takes on a chronic fibroid type. There is nothing in the symptoms which characterise a tubercular endometritis from any other form, though the family history or the occurrence of other tubercular foci may point to this. Examination of particles of endometrium removed by the curette may be made for tubercle bacilli in suspicious cases.

Tubercular infection of the Fallopian tubes is the most frequent form of tuberculous disease of the genital tract. Hegar, Martin,[2] Orthmann, and Whitridge-Williams[3] have done much to add to our knowledge of this subject, the last observer maintaining that the majority of cases are secondary to tuberculosis elsewhere, and are either due to infection from the blood or the neighbouring organs. Doran also thinks the same.[4]

Primary disease of the tubes is possible, but great care must be taken to exclude other possible tubercular lesions present.

It is of extreme interest that tubercle of the tubes may occur in young children and virgins.[5] The tubes are sometimes found studded with miliary tubercles, and may be little altered in shape, or large ovoid banana-shaped swellings occur, with thick walls, the interior of which may be ulcerated and the cavity filled with curdy pus.

Tubercular salpingitis has already been described in the chapter on Diseases of the Fallopian Tubes. According to some observers it would seem probable that a pre-existing gonorrhœal salpingitis would predispose the tube to a tuberculous infection.[6]

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[1] Reed, *op. cit.* p. 390.

[2] *Die Krankheiten der Eileiter*, 1895.

[3] Whitridge-Williams, 'Tuberculosis of the Female Generative Organs,' *Johns Hopkins Hosp. Rep.* vol. iii. 1892; Doran, art. 'Diseases of Fallopian Tubes,' Albutt and Playfair, *System of Gynæcology*, p. 793.

[4] Doran, *op. cit.* p. 795.

[5] *Ibid.* p. 793.

[6] Reed, *op. cit.* p. 526.

Tuberculosis of the ovary has been observed,[1] and may occur with a general tubercular peritonitis or salpingitis. Primary tuberculosis of the ovary is very rare. Tubercle of the peritoneum[2] in women may occur in the acute miliary or the caseating form, or even as a chronic fibro-tuberculosis.

It may arise by extension from the Fallopian tube, or other neighbouring organs, or may be part of a blood infection.

It is characterised by the development of ascites, tumour formation, or caseous abscesses.

The adhesion of the various viscera of the abdomen to one another may be very pronounced, forming large masses, or the omentum, mesentery, and bowel may enclose fluid which simulates an ovarian cyst.

Tubercular peritonitis occurs at all ages, but the incidence is most frequent between twenty and forty.[3] Osler states that the laparotomy cases he has collected are twice as numerous in females as in males. A peculiar feature of the disease is the uncommon occurrence of grave tuberculous lesions in other parts of the body.[4]

The bladder[5] may be affected either primarily or as a descending infection from tuberculous disease of the kidneys, or as a part of general tuberculosis. It may be accompanied by the most troublesome and distressing cystitis.[6] Tubercle of the bladder is stated by Ultzmann[7] to be sometimes associated with gonococcus infection. Tuberculosis of the kidneys and ureters is described in works on Surgical Pathology.

Bacillus capsulatus aërogenes[8] (fig. 148).—This organism was described originally by Welch and Nuttall as occurring in the blood-vessels *post mortem*, and has been found in the uterine vessels of women during septicæmia after confinement or after abortion cases, and in emphysematous gangrene.[1] It is the most frequent cause of so-called post-mortem emphysema.[2]

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[1] Reed, *op. cit.* pp. 575, 576.

[2] Osler, *op. cit.* 1898 ed. p. 286; Reed, *op. cit.* p. 692.

[3] Osler, *op. cit.* p. 237.

[4] Reed, *op. cit.* p. 692.

[5] Herman, *Diseases of Women*, p. 676; Bowlby, *Surgical Pathology*, 4th ed. 1900, p. 455.

[6] Howard Kelly, *op. cit.* vol. i. p. 376.

[7] Ultzmann, *Die Krankheiten der Harnblase*, Stuttgart, 1890, p. 151.

[8] Welch and Nuttall, *Johns Hopkins Hospital Bulletin*, July, August, 1892; see also Welch and Flexner, 'Observations on the Bacillus capsulatus aërogenes,' *Journ. of Experimental Medicine*, Jan. 1896, vol. i. p. 5; Reed, *op. cit.* p. 54; Sternberg, *Manual of Bacteriology*, 1893, p. 731; Howard Kelly, *op. cit.* vol. i. p. 40; McWeeny, *Lancet*, vol. i. 1901, p. 101; Lindenthal, 'Zur Etiologie der sogenannten Kolpohyperplasia cystica,' *Wiener Klin. Wochenschr.* 1897, No. 1; Graham, Steward, and Baldwin, *Columbus Med. Journ.*, August 1893.

The bacillus is probably a constant inhabitant of the intestines, and probably also of the superficial layers of ordinary soil.

The evidence that it can by itself cause an infection in man *intra vitam* is not conclusive. In the majority of such cases other organisms have been found associated with it. In other cases it appears to have occurred as a terminal infection in patients dying from other causes.

It is a strict anaërobe. It is a large, straight bacillus, with rounded ends, from 3μ to 6μ in length, growing singly or in chains of three or four. It has a distinct and very characteristic capsule, and generates gas in large quantities in all culture media. It may be responsible for the gas which occasionally occurs in tissues in connection with suppuration.[3]

Lindenthal[4] thinks that some cases of so-called kolpohyperplasia cystica, or vaginitis with gas cysts, may be due to an organism which he calls the bacillus emphysematis vaginæ. It is probably identical with the bacillus capsulatus aërogenes. Herman has collected the literature on this subject.[5]

Some curious cases of physometra in pregnancy,[6] or in the puerperium where the uterus is distended with fœtid gas, are probably of this description. It is, however, possible, as before stated, that in such cases the infection may be a terminal one. Physometra may also accompany pyometra, or sloughing uterine fibroids.

SAPRÆMIA, SEPTICÆMIA, AND PYÆMIA

Sapræmia is generally understood to mean the constitutional condition resulting from the absorption of the chemical

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[1] Eugen Fränkel, *Ueber Gasphlegmonen*, 1893.

[2] See also specimen, St. Bartholomew's Hospital Museum, 3382K, solid ovarian tumour with post-mortem gas cysts.

[3] Reed, *op. cit.* p. 55.

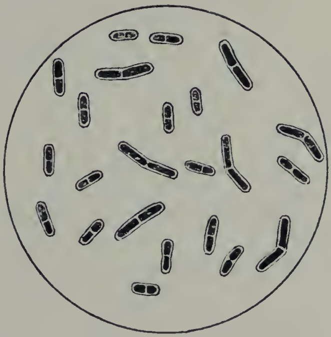
[4] Lindenthal, *op. cit.*

[5] Herman, *Diseases of Women*, p. 441; *id. Lancet*, vol i. 1891.

[6] Howard Kelly, *op. cit.* vol. i. p. 555; also Dobbin, 'Puerperal Sepsis due to infection with Bacillus capsulatus aërogenes,' *Johns Hopkins Hospital Bulletin*, Feb. 1897.

Fig. 148.

BACILLUS CAPSULATUS AËROGENES.



Semi-composite drawing from a pure culture, modified from Howard Kelly (after Welch and Nuttall). (High power.)

products of pyogenic or septic organisms,[1] the organisms themselves not having invaded the living tissues.

For want of a better word we give the name 'toxine' to such chemical substances. Our knowledge of the chemical nature of the metabolic products of micro-organisms is very imperfect. Kanthack[2] says that 'while we are still unacquainted with the chemistry of the tissues, while proteids, peptones, or albumoses are little more than names, it is almost useless to speculate on the chemical changes induced by micro-organisms in living tissues. In artificial media they either take up certain substances or elements, thereby splitting up complex molecules, or atomic groups, thus altering the original medium in which they were placed; or, on the other hand, they may secrete complex substances which they have built up from simpler assimilated bodies.'

If fluid be taken from decomposing material and the organisms filtered off, and the bacteria-free filtrate then injected into an animal, the latter manifestly suffers, not from the micro-organisms themselves, but from the chemical products of their action.

To the condition which results the name *sapræmia* or septic intoxication is applied.[3] The effects depend on the dose of poison; it may be large enough to kill, or recovery may take place.

The best possible example of this occurs in the uterus which has not been properly emptied after delivery, or which contains clots, devitalised decidual débris, or stagnant lochia.

Micro-organisms, once implanted on such a soil, grow and multiply, producing toxines which are absorbed into the circulation, producing sapræmia.

In these cases, the organisms themselves do not multiply in the healthy living tissues of the part, but are restricted to the dead or dying material which has been retained. Remove the dead tissues, the clot or putrid placental mass, and the source of poison and the micro-organisms are removed too, and the patient recovers. If the dead tissues be left, the processes of decomposition, necrosis, and toxine formation continue. Hence in sapræmia the symptoms are always proportional to the dose and virulence of the poison absorbed.

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[1] Bowlby, *op. cit.* 4th ed. p. 74. The organisms being saprophytes and the common organisms of decomposition, some are bacilli, others belong to the proteus group.

[2] Kanthack, Bowlby's *Surg. Pathol.* 4th ed. pp. 22–25; Muir and Ritchie, *op. cit.* 2nd ed. pp. 144–162.

[3] Bowlby, *op. cit.* 4th ed. p. 74. See also table, *ibid.* p. 85.

Of course, the organisms themselves which produce the poison can be transmitted from one individual to another, or to another suitable surface, *e.g.* to another puerperal uterus, by carelessness, or from want of cleanliness, and produce similar effects.

It is out of place here, perhaps, to speak of the so-called puerperal fevers, and the subject is only referred to shortly, though its importance is very great indeed.

Puerperal fever is but a name; there is no such thing apart from the ordinary septic processes; and when we speak of puerperal fever we mean sapræmia or septicæmia which is affecting the lying-in woman. As such, these septic conditions are the result of infection from without, and in all, contact is the danger, and transference of septic organisms from one breeding-ground to another is 'the' thing against which we have to be on our guard.[1]

The theories of auto-infection in such cases have long ago been given up.

Septicæmia.—This differs from sapræmia in that the micro-organisms are capable of living and multiplying in the living tissues and blood, thus producing a general or local infection. Therefore, in septicæmia the bacteria are parasites growing on the living tissues of the host, as compared with the organisms of sapræmia which grow as saprophytes on dead material.

Strictly speaking,[2] the term septicæmia should be applied only to those cases in which there is an infection of the blood-stream, but clinically it includes many cases where the blood-stream remains free from organisms.

In this wide sense the disease may in some cases be localised, the micro-organisms remaining at the original seat of inoculation, or at the most spreading only by direct extension;

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[1] Herman, 'Prevention of Puerperal Fever in Private Practice,' *Brit. Med. Journ.* Jan. 11, 1896; Cullingworth 'On the Undiminished Mortality from Puerperal Fever in England and Wales,' *Brit. Med. Journ.* vol. i. 1897, p. 573; *Trans. Obstet. Soc. Lond.* vol. xxxix. 1897, p. 91; Boxall, 'Fever in Child-bed,' *Trans. Obstet. Soc. Lond.* vol. xxxii. 1890, p. 219, and *id. Lancet*, July 1, 1893.

[2] Bowlby, *op. cit.* 4th ed. p. 77.

in others, the bacteria may enter the lymphatics, or blood-stream, or both, and be carried to other parts where they may live and multiply.

Though in cases of septicæmia the bacteria are living and multiplying in the living tissues of the host, the conclusion must not be drawn that the symptoms are wholly or to any great extent the result of their mere mechanical action. The symptoms, fever, rigors, malaise, &c., are due to the absorption of chemical poisons produced by the bacteria. To this by no means small extent, therefore, septicæmia is an intoxication, and it is to be the more feared than sapræmia only, because the organisms, having spread beyond the original seat of inoculation, cannot be in most cases removed from the body by any operative procedure.

Though the bacteria cannot be removed, the local or general resistance of the tissues of the patient may be great enough to overcome the infection ; if so, the patient recovers.

A large proportion of cases of septicæmia are due to streptococcus infection. It is possible also that septicæmia and sapræmia may co-exist.

It is noteworthy that in septicæmia there is sometimes one severe initial rigor, which is not repeated, as in sapræmia, but this is by no means always the case ; in fact, in ulcerative endocarditis frequent rigors are common.

The outlook in septicæmia is always serious ; in severe cases death may occur within twenty-four hours, and in fatal cases life is rarely prolonged for more than seven or eight days. The results of the use of antistreptococcus serum in such cases have been disappointing. It is possible that the number of species of pathogenic streptococci is larger than has been supposed, and it is probable that a given serum can avail only against infection by the particular species of streptococcus which has been used in its preparation.[1] On post-mortem examination there may be no focal lesions in the viscera, and the seat of infection may present only slight changes. The spleen is enlarged and soft, the blood may be extremely dark in colour, and hæmorrhages are common, particularly on the serous surfaces. Neither thrombi nor emboli are found.

Pyæmia.—This is a form of septicæmia characterised by the formation of secondary metastatic inflammation, and is a general disease accompanied by recurring rigors and intermittent fever, which results from the infection of the blood by the bacteria of suppuration and their products.[1]

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[1] Bowlby, *op. cit.* 4th ed. p. 86.

The micro-organisms in pyæmia are the same as those which produce septicæmia; the seat of inoculation is generally of recent origin;[2] wounds on surfaces which are granulating are rarely infected. In connection with puerperal sepsis, the placental site, or wounds of the cervix, vagina, vulva, and perineum may be the seat of infection.

The most rapidly fatal cases of pyæmia survive longer than do those of septicæmia.

The most frequent complications are broncho-pneumonia and pleurisy, pericarditis and endocarditis, mutiple abscesses, especially of the lungs, painful swellings and suppuration of joints, peritonitis, and thrombosis of the veins in the neighbourhood of the seat of inoculation.[3]

The veins, especially those of the broad ligament, in uterine pyæmia, may be found filled with clot, and this may extend for some considerable distance. The clot is generally friable and disintegrating, and even true pus may occur.

Commonly, the transmission to various parts from the local focus takes place by the fragments of the thrombi, which are carried as emboli in the blood-stream to distant parts. The streptococcus pyogenes is most frequently found in the pus at the primary seat of infection, and in the metastatic abscesses.[4]

Not only are emboli formed, but the embolus itself is infective; it contains micrococci identical with those contained in the clot in the vein from which it was derived. These independent suppurative centres are typical of pyæmia, and may occur in the lungs, heart, or kidneys; while foci in the region of the portal system produce abscesses of the liver, with or without suppurative pylephlebitis. A very interesting variety of pyæmia is seen in malignant endocarditis.

The symptoms in pyæmia are in the first place due to the intoxication, but in part also to the mechanical effects of embolism, and rigors may be frequent.

It is difficult to find special names for all the various septic processes, but pyæmia as a clinical term can only be applied to a septicæmia accompanied by metastatic infective embolism.[1] *Post mortem*, the muscles are frequently more or less stained as in septicæmia, and the pleuræ often contain foul blood-stained fluid mixed with pus and fibrin. Similar fluid may be found in the pericardium, which may be covered with recent lymph. Raised patches, varying in size from a pea to a walnut, may be seen beneath the pleural surface; they consist of congested areas of lung tissue frequently containing pus. Broncho-pneumonia is common, and there may be pus in the smaller tubes. No abscesses are, however, usually found in the lung itself; they are confined to the sub-pleural tissue.[2]

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[1] Osler, *op. cit.* 1st ed. p. 115.

[2] Bowlby, *op. cit.* 4th ed. p. 79.

[3] *Ibid.* p. 80.

[4] Osler, *op. cit.* 1st ed. p. 116.

Vessels in the thrombosed areas contain on microscopic examination numerous micro-cocci, which may even completely plug the smaller capillaries.

Endocardial inflammation, or small abscesses in the heart muscle, may be found.

The peritoneum may contain fluid similar to that met with in the pleural cavities and pericardium.

The spleen is large, soft, and friable, as is also the liver; while both these and the kidneys may contain abscesses.

The joints may contain thin oily pus, and the cartilages may, or may not, be destroyed.[3]

Parotid abscess is not uncommon.[4] Examination of the wound or placental site may show retained pus, or a sloughy gangrenous condition. In the neighbourhood, at all events in uterine pyæmia, the vessels are plugged, and the clotting may extend to the vessels of the broad ligament, or even the iliac and femoral veins. The clot is always very ill-formed and friable, and may be suppurating. The vessel walls are stained, or roughened and ulcerated.

Frequently in uterine pyæmia, especially associated with wounds of the uterus, the cellular tissue of the broad ligaments is extensively involved, and a large phlegmon results, amid which are many areas of abscess formation, so that the tissues look like a coarse sponge.[5] Matthews Duncan has pointed out that in many cases of puerperal septicæmia or pyæmia such phlegmonous masses, as distinguished from abscesses, are not rare.[1] They may occur on the limbs, and are sometimes multiple.

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[1] Bowlby, *op. cit.* 4th ed. p. 84. [2] *Ibid.* p. 80. [3] *Ibid.* p. 81.

[4] Parotitis after abdominal section is not uncommon even in cases apparently not septic. Grieg Smith, *Abdominal Surgery*, 4th ed. p. 100.

[5] Lewers, 'Post-mortem Appearances of a Phlegmon of the Broad Ligament,' *Trans. Obstet. Soc. Lond.* vol. xxx. 1888, p. 7.

The author believes that some of the curious brawny swellings of the vulva, perineum, and buttocks in similar cases are of this nature; while some forms of so-called white leg are essentially septic.[2]

Is there a Parasite of Cancer? (Figs. 149, 150, 151.)—The subject has already been touched upon in the chapter on Cancer of the uterus, and is certainly of great pathological interest; but further investigations seem necessary before any definite conclusion can be arrived at.

It is of course possible that 'parasites' exist, at all events in one special group of tumours, the carcinomata; but even in this limited field it is not by any means proved to be always the case.[3]

Lately much attention has been paid to this matter in order to demonstrate the presence of parasites, though it is now many years since the idea first arose that cancers might be due to some micro-organism.[4]

Nepveu, Rappin, and Scheuerlen[5] have described special bacilli, but little attention was paid to the subject till after 1892, when Thoma, Malassez, Dorier, Wickham, Ruffer and Walker,[6] and others, described so-called parasitic organisms in cancer.[7] Attempts at cultivation met with negative results. Most of the observers who accepted the parasitic nature of these cell inclusions were inclined to refer them to a branch of the protozoa, viz. the sporozoa or psorosperms; others regarded them as yeasts.[8] Coccidia are parasitic in many animals, and in rabbits one variety may give rise to tumours of the liver, but these tumours do not resemble carcinomata. Psorosperms are also found in Paget's disease of the nipple.

Russell[9] has described bodies in cancer cells, demonstrated

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[1] Matthews Duncan, *ibid.* p. 9.

[2] Roberts, 'On the Common Form of White Leg after Confinement,' *Trans. Obstet. Soc. Lond.* vol. xxxvii. 1895, p. 163.

[3] Montague Murray, *Green's Pathol.* 9th ed. p. 77.

[4] 'The Etiology of Cancer,' *Lancet*, vol. i. 1901, p. 1413.

[5] See H. G. Plimmer, *Practitioner*, May 1899, p. 432.

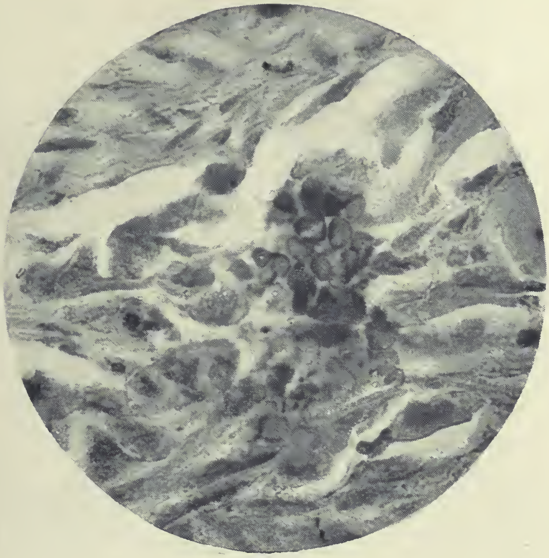
[6] Ruffer and Walker, *Brit. Med. Journ.* vol. ii. 1892, p. 113; *iid. Journ. of Pathol.* Oct. 1892.

[7] See references in Metchnikoff's paper, *Brit. Med. Journ.* vol. ii. 1892, p. 127.

[8] *Lancet*, *loc. cit.* p. 1413.

[9] Russell, 'An Address on the Characteristic Organism of Cancer,' *Brit. Med. Journ.* vol. ii. 1890, p. 1356.

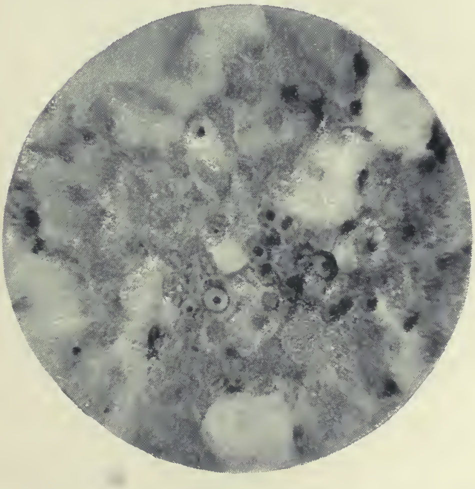
Fig. 149.
PARASITES IN CANCER.



Section of cancer of the breast, to show group of parasites in the centre of the field. (× 350. Keith W. Montserrat.)

(Mitchell Banks, 'Lancet,' vol. i. 1900, p. 997.)

Fig. 150.
PARASITES IN CANCER.

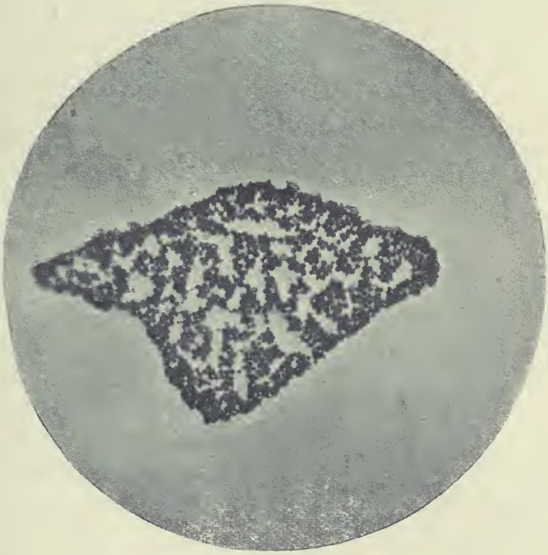


Section of carcinoma of the breast, to show the cancer parasites. Two in the centre are well defined. (Reichert, $\frac{1}{15}$-inch oil immersion. R. I. M. Buchanan, × 375.)

(Mitchell Banks, 'Lancet,' vol. i. 1900, p. 997.)

Fig. 151.

PARASITES IN CANCER.



Coverslip preparation of parasites cultivated on artificial media, from a mammary carcinoma × 75. (Low power. Keith W. Montserrat.)

(Mitchell Banks, 'Lancet,' vol. i. 1900, p. 997.)

by special staining methods, which he called 'fuchsin bodies' on account of their special affinity for that particular stain. He considered them to belong to the yeast group.

Soudakewitch[1] and Metchnikoff[2] have also written on this subject.

Sanfelice[3] was of the opinion that the so-called parasitic organisms were yeasts.

Plimmer[4] described a new method of staining these bodies. His statements were based on the examination of 1,278 specimens of cancer; in 1,130 of these he was able to demonstrate bodies which he considered to be parasites. Out of these cases 165 were cancers of the uterus and vagina.

Mitchell Banks[5] refers to the subject in the 'Lettsomian Lectures on Cancer of the Breast' for 1890.

Sjöbring[6] agrees with Metchnikoff that the organism is of an animal nature, and records a case where intra-peritoneal inoculation in a white mouse of a culture from a colloid cystoma of the ovary produced a similar growth of small colloid cysts, lined by epithelium.

Further researches have been undertaken by Olt,[7] Schüller,[8] Shattock and Ballance,[9] Klein,[10] Soudakewitch,[11] Foà,[12] Busse,[13] and others, on this subject.

In America, Nichols, Greenough, and Richardson report negative results, at all events with the experimental inoculation and cultivation of the blastomyces of Sanfelice and Plimmer.[1]

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[1] Soudakewitch, *Annales de l'Institut Pasteur*, March 1892, p. 145, and *ibid.* August, p. 545; also *Brit. Med. Journ.* vol. ii. 1892, p. 1275.

[2] Metchnikoff, 'Remarks on Carcinoma and Coccidia,' *Brit. Med. Journ.* vol. ii 1892, p. 1273.

[3] *Lancet*, *loc. cit.* p. 1413; Sanfelice, *Zeits. f. Hygiene und Infections-Krankheiten*, vol. xxix. 1898; see also *Practitioner*, *loc. cit.* p. 433.

[4] Plimmer, *Practitioner*, April 1899, p. 433, where other articles on this subject may be found; see also *Brit. Med. Journ.* vol. ii. 1892, p. 1277.

[5] Sir W. M. Banks, *Lancet*, vol. i. 1900, pp. 684, 843, 990. The author begs to thank Sir W. M. Banks for allowing him to reproduce the microphotographs, figs. 149, 150, 151.

[6] Sjöbring, *Fortschritte der Medicin*, 1890, p. 529, and *Brit. Med. Journ.* vol. ii. 1890, p. 1519.

[7] Olt, *Deutsche Tierarzt-Wochenschrift*, 1900, Nos. 22, 23; *Brit. Med. Journ.* voi. i. 1901, p. 357.

[8] Schüller, *Brit. Med. Journ.* vol. ii. 1900, p. 1518.

[9] Shattock and Ballance, *Brit. Med. Journ.* vol. i. 1890, p. 565.

[10] Klein, *Beiträge zur Path. Anat.*, 1891, vol. xi.

[11] Soudakewitch, *op. cit.* and *Brit. Med. Journ.* vol. ii. 1892, p. 1275.

[12] Foà, *Gazzetta di Medicina di Torino*, 1891, No. 36.

[13] Busse, 'Ueber Saccharomycosis Hominis,' *Virchow's Arch.* vol. cxl. 1895, p. 23.

Gaylord[2] has published the results of his investigations on this subject in the New York State Pathological Laboratory of the University of Buffalo, and appears to be satisfied that the organism is a protozoon and not a yeast.

The organisms were present in all forms of rapidly growing tumours, and the fluid of malignant ovarian cysts (adeno-carcinomata) was found to contain a large number of these bodies, and Gaylord was led to the conclusion that all the organs, including the blood taken from all regions of all cases dying of cancer, including sarcoma and epithelioma, contain large numbers of these organisms.[3]

Perhaps at the present time, though the subject is of great interest, it is unwise to express any definite opinion on the question of a parasite of cancer, for more proofs must be obtained that such bodies are parasitic, and further, that they are the cause of the new growth in which they are found.[4] Most observers hold that these parasites are only masses of hyaline material, fragmented nuclei, leucocytes, or invaginated cells.[5]

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[1] See 'Cancer Investigation in America,' *Brit. Med. Journ.* vol. i. 1900, p. 358.

[2] Gaylord, 'The Protozoon of Cancer,' *Amer. Journ. of Med. Sciences*, May 1901, vol. cxxi. No. 349, p. 503; also *Lancet*, leading article, 'The Etiology of Cancer,' vol. i. 1901, p. 1413.

[3] Gaylord, *op. cit.* p. 513–516. Several varieties of the so-called organisms are described by Gaylord. Some are small highly refractile bodies, others are larger and may throw out pseudopodia; while saccular forms exist which contain refractile bodies like spores.

[4] Bowlby, *Surg. Pathol.* 4th ed. p. 160; Kanthack, *ibid.* p. 13.

[5] Green's *Pathology*, 9th ed. 1900, p. 77.

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